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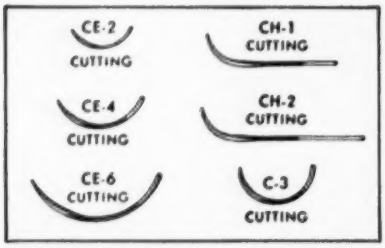

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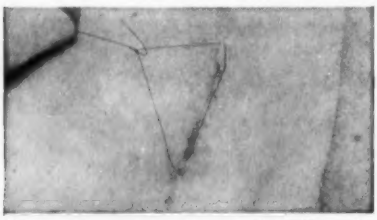

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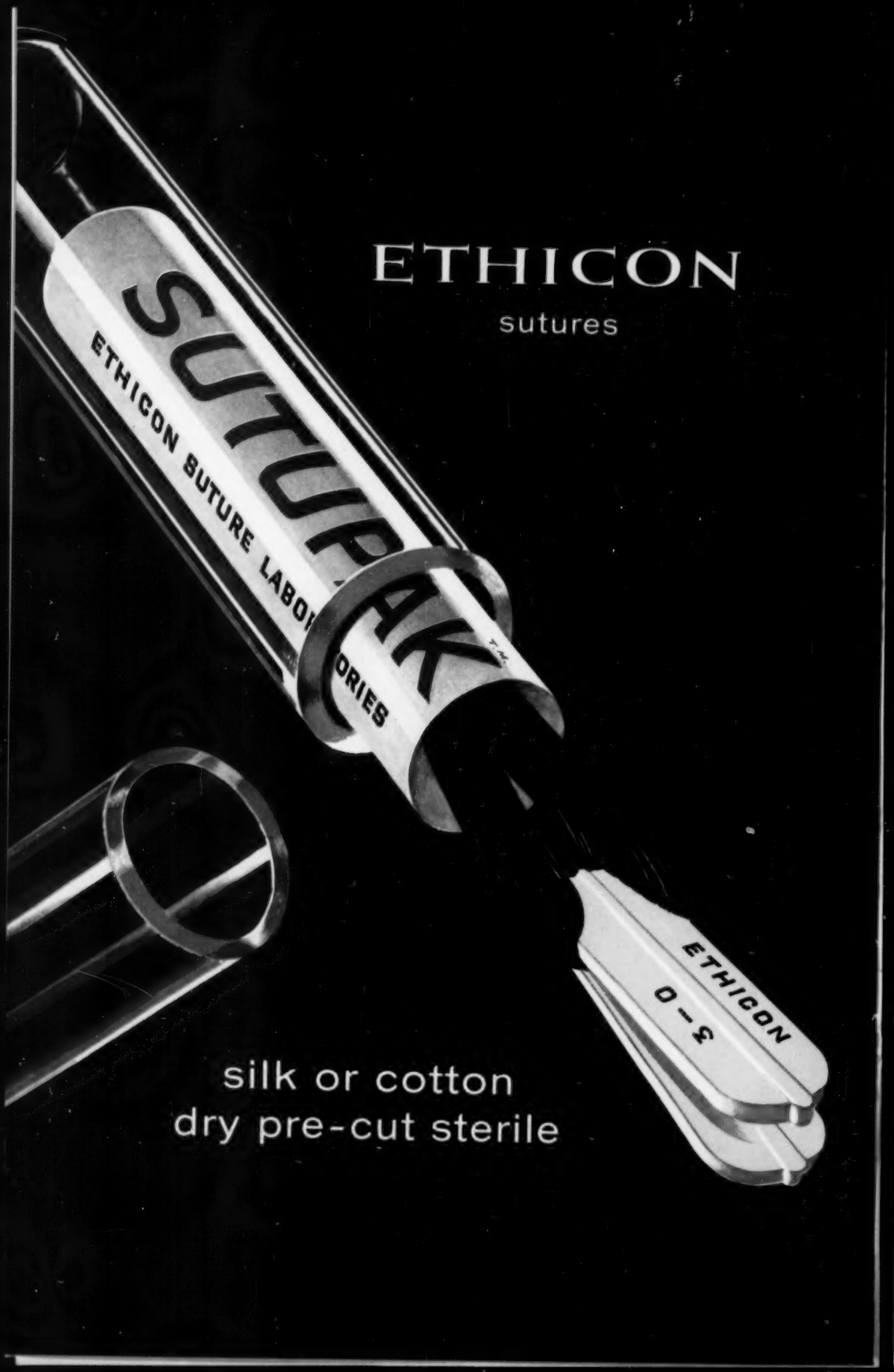
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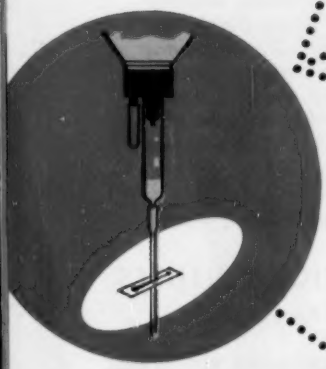

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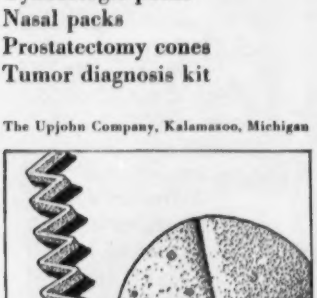

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VOLUME 68 MARCH 1954 NUMBER 3

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Editorials

UNNECESSARY SURGERY AND INADEQUATE SURGERY

IN THE continuing program of the American College of Surgeons to raise the standards of surgical care, much rewarding effort has been expended on the problem of reducing to a minimum unnecessary surgery. One method among others has been the formation in each hospital of a Tissue Committee, whose function it is to scrutinize all surgical specimens in order to estimate the extent, if any, of unnecessary surgery. These Tissue Committees could also use the same material to study the problem of inadequate surgery.

Adequate surgery is, of course, particularly essential in many kinds of cancer in order to give the patients their best chance for survival. In certain fields there are honest and justifiable differences of opinion as to what would constitute adequate surgery, particularly in terms of the recent trend toward more radical procedures. On the other hand, in many cases there is well-established evidence to support readily stated criteria for the adequate removal of malignant disease. The surgeon who in such a case fails to do the full operation should be required to justify the inadequate procedure and explain the extenuating circumstances, if any, which influenced his decision.

Carcinoma of the breast is a case in point, in view of two recent studies containing data regarding inadequate surgery in this disease. In one report,[1] by Monroe, a series of patients were followed for five years or longer after radical mastectomy by 15 different surgeons. The lymph nodes were carefully counted in each specimen after it arrived at the laboratory. A wide difference was found in the number removed by the different operators. At least two of the surgeons were apparently content with the removal of relatively few lymph nodes, whereas the others consistently removed a much greater number. The five-year follow-up results were startling and significant. Of the patients operated upon by two surgeons who removed specimens containing an average of 15 and 28.2 lymph nodes respectively, only 50 and 66% of the patients were alive at the end of five years, even though none of the lymph nodes in the specimens showed carcinoma. By contrast, of the patients operated on by four surgeons whose specimens contained an average of 28.8, 35.8, 38 and 46.3 lymph nodes respectively (none containing cancer) all were alive at the end of five years.

A similar study from another part of the country was carried out by Sprong and Pollock.[2] In a group of patients without node metastases, those operated upon by four surgeons whose specimens contained an average of 28.8 or more lymph nodes, all were alive at the end of five years, whereas of the patients operated on by one surgeon whose specimens contained an average of 16 lymph nodes, only

half were alive at five years. It is likely that similar studies in other hospitals would reveal similar or more striking differences. There are, of course, many factors involved in life expectancy following cancer surgery. Moreover, the number of lymph nodes available for removal certainly varies. Nevertheless, it seems clear that other things being equal, these studies show that the number of lymph nodes removed plays a decisive part in the prognosis of cancer of the breast. Actually, these observations merely add confirmation to the principle known for over 60 years of the importance of a careful and extensive axillary dissection in cancer surgery of the breast. They also show that counting lymph nodes is one available method of measuring the adequacy of the operation.

What is shocking, therefore, is to realize that patients with cancer of the breast are still being subjected to an inadequate operation which can only shorten their maximum span of life and thus needlessly increase cancer mortality. In the strenuous drive against cancer, this aspect of therapy must be emphasized more widely, especially since the breast ranks so high as a frequent site of cancer. It seems wasteful and myopic to expend so much effort on programs to detect cancer early without at the same time taking measures to insure its adequate removal. An important part of the responsibility of the Tissue Committees as recommended by the American College of Surgeons should be the assay of adequate surgery as well as of unnecessary surgery. Even if time-consuming techniques of examining specimens are required to make such studies, the potential rewards to the patient are so great as to make the effort worth while.

ROBERT ELMAN, M.D.

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1. Monroe, C. W.: A. M. A. Arch Surg. **57**:479, 1951.
2. Sprong, D. H., and Pollock, W. F.: Ann. Surg. **133**:330, 1951.

CONGENITAL PYLORIC STENOSIS AT THE BOSTON CITY HOSPITAL

MARTIN J. ENGLISH, M.D.
JOHN H. CRANDON, M.D.
MAX RITVO, M.D.
AND
GERARD DESFORGES, M.D.
BOSTON

IN A LARGE general hospital with an insufficient number of pediatric surgical cases to warrant a full-time specialist in this field, the policy with reference to surgery in children may be somewhat problematical. Should pediatrics surgery be performed by all general surgeons or should it be on an assignment basis? The ordinary run of appendectomies and herniorrhaphies in children would seem to be adequately handled by the average general surgeon. However, in conditions where the preoperative and postoperative care is more involved, even though the surgery be uncomplicated, the average surgical service may find the ground so unfamiliar that suboptimal conditions prevail. A common condition of infants exemplifying the questions involved is congenital hypertrophic pyloric stenosis.

Prior to Dec. 1, 1949, the surgery of children at the Boston City Hospital was performed by several different general surgeons on two different surgical services. At this time, in an effort to improve the mortality rate of pyloric stenosis, the following policy was adopted: 1. All patients were operated upon by one team of surgeons. 2. Each infant remained on the pediatrics service throughout his hospital stay. 3. Effort was made to establish the diagnosis more quickly and thereby bring the infant to surgery sooner.

During the following three years, Dec. 1, 1949, through Dec. 1, 1952, there were 41 cases of congenital hypertrophic pyloric stenosis, either confirmed or suspected, in which operation was done, and these are the subject of this report. There were no deaths. The surgical aspects of this report are presented from the viewpoint of a general rather than a pediatric surgeon.

From the Pediatrics and First Surgical Services, Boston City Hospital.

Formerly Pediatrician-in-Chief, Boston City Hospital; Professor of Pediatrics Emeritus, Boston University School of Medicine (Dr. English).

Junior Visiting Surgeon, Boston City Hospital; Assistant Professor of Surgery, Tufts College Medical School (Dr. Crandon).

Radiologist-in-Chief, Boston City Hospital; Assistant Professor of Radiology, Harvard Medical School (Dr. Ritvo).

Resident, Thoracic Surgical Service, Boston City Hospital; formerly Resident, First Surgical (Tufts Teaching) Service, Boston City Hospital (Dr. Desforges).

ETIOLOGY

To us, the etiology of this condition remains obscure. In our series true pyloric tumors were found in patients 3, 4, and 5 days of age, respectively, indicating that the condition may occasionally, at least, be present at birth.

PATHOLOGY

The pathological characteristics of the pyloric mass have been well described.* In this series, in all but five cases there was the usual olive-sized ovoid tumor of cartilaginous consistency. Three patients with conditions clinically indistinguishable from pylorospasm showed questionable muscular hypertrophy at operation. In two other patients falling in the same clinical category, the pylorus appeared normal at operation. In all of these cases the Ramstedt procedure was nevertheless performed.

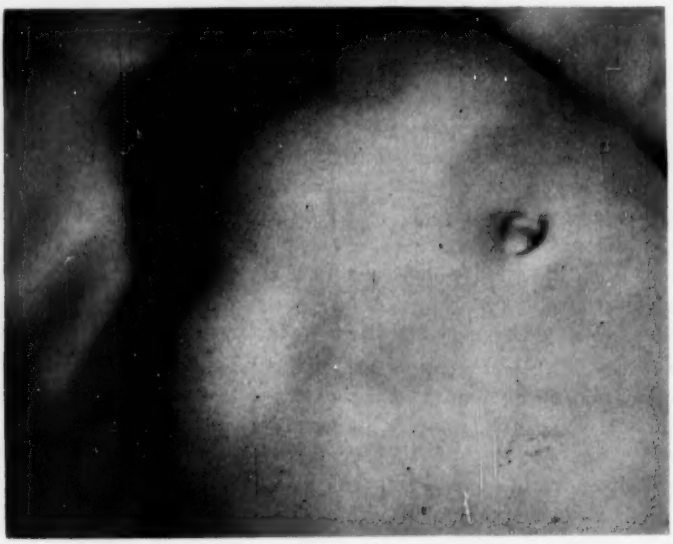

Fig. 1.—Gastric peristaltic waves following feeding.

DIAGNOSIS

Generally the diagnosis of congenital hypertrophic pyloric stenosis is easily made, but occasionally it has been difficult to confirm, in our experience. The characteristic picture of high intestinal obstruction with visible peristaltic waves (Fig. 1), projectile vomiting, and rapid loss of fluid and electrolytes is well known. It has been stated that the vomiting does not commence until after the first 10 days to two weeks of life, but in three patients of this series it began at birth. At first there may seem to be merely regurgitation of small amounts of each feeding. Gradually, however, frank vomiting develops, which soon becomes projectile in character. The absence of bile in the vomitus is an important diagnostic criterion.

* References 1 and 2.

A desire to nurse and take the bottle with seeming relish following a bout of violent vomiting is again in favor of this diagnosis. With the continuous vomiting, scanty stools, and starvation, the signs of severe dehydration and alkalosis appear.†

In the differential diagnosis of congenital hypertrophic pyloric stenosis certain other causes of vomiting must be considered. Intracranial injury may be suspected, particularly in the immediate neonatal period. However, in this condition, vomiting is generally not constantly related to feeding, neurological signs frequently may be elicited, and the lumbar puncture may be extremely helpful. Obscure infectious processes, such as otitis media, must be kept in mind as a cause of vomiting. Atresia, stenosis, or malrotation of the gastrointestinal tract may cause difficulty, but their

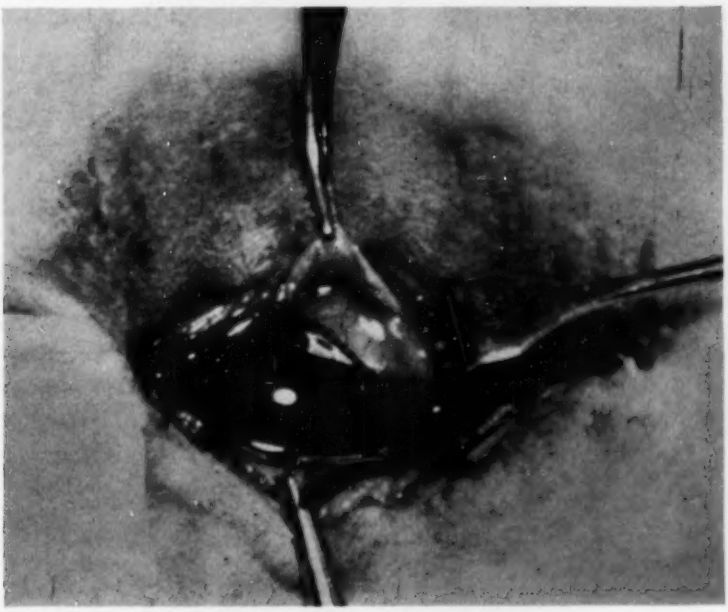

Fig. 2.—Upper right rectus incision. Presenting liver edge showed subcapsular hemorrhage, presumably from trauma attendent upon attempts to palpate tumor lying underneath.

presence is indicated from birth by bile-stained or fecal vomiting and by varying degrees of distention. Improper feedings, such as too concentrated formulae or prolonged breast suckling, may lead to vomiting. In our experience, the most difficult and the commonest problem of differential diagnosis is the distinction of congenital hypertrophic pyloric stenosis from pylorospasm. In the latter condition there is generally an intermittent pattern of vomiting. The vomitus usually contains some bile, and the symptoms may be relieved totally or in part by atropine derivatives. Frequently, however, neither of these axioms obtain. In this series of patients complete refractoriness of pylorospasm to medical management was considered an indication for surgical intervention.

† References 3 to 7.

On physical examination palpation of an olive-like tumor in the right upper quadrant or epigastrium established the diagnosis of congenital hypertrophic pyloric stenosis. Attempts at palpation are most fruitful immediately after vomiting, since the abdominal wall then is relaxed. Although most authorities ‡ state that the pyloric mass can be felt in 90 to 95% of cases, unequivocal findings on palpation were present in only about one-half the cases of this series. It is our impression, therefore, that those who see only an occasional patient with this condition should

Fig. 3.—Roentgenogram showing "string sign," occasionally seen with barium meal.

not be dismayed at inability to feel the tumor, nor should they defer operation until the tumor is palpable, when numerous other positive findings point to this diagnosis. In two patients of this series repeated attempts to palpate the tumor resulted in a finding of subcapsular hematoma of the liver at operation (Fig. 2).

If, after evaluation of all findings, the diagnosis is still in doubt, it is not harmful, in our experience, to seek more information by x-ray studies of the upper gastrointestinal tract. This form of examination was utilized in 23 of our 41 cases without a single untoward result.

‡ References 1, 2, 4, and 8.

Prior to administration of the opaque meal, which should be a thin barium-water mixture, a plain roentgenogram of the abdomen should always be made to rule out the presence of dilatation or other demonstrable abnormalities of the stomach. If dilatation is present, it can be overcome by insertion of a nasal catheter. Roentgen observation should commence with the ingestion of the opaque medium in order to determine the initial emptying time. This in itself may suffice to demonstrate that congenital hypertrophic pyloric stenosis is present, since elongation of the pyloric canal, and/or the so-called "string sign" as manifested by a linear streak of barium at the region of the pylorus, may be observed (Fig. 3). Demonstration by x-ray of an elongated and narrowed pylorus is pathognomonic of congenital hypertrophic pyloric stenosis. Frequently, however, the ingested barium mixture at first passes through a relatively normal-appearing pylorus at a more or less normal rate.

Fig. 4.—Barium meal at end of four hours. Pylorospasm and cardiospasm. Retained barium in esophagus may be seen just to the right of the pylorus.

A very significant finding by x-ray is the presence of a smooth, round, symmetrical filling defect, caused by narrowing of the prepyloric segment. Of significance also is a rounded area of radiance at the base of the duodenal bulb, which results from protrusion of the hypertrophied muscle mass into the duodenum. Most commonly in this series the outstanding finding by x-ray was the markedly delayed gastric emptying, with retention of large amounts of barium in the stomach at 6, 12, and 24 hours.

The presence of a relatively normal upper gastrointestinal series in the presence of obstructive symptoms does not preclude the possibility of pyloric stenosis, since this occurred in three patients two weeks, one week, and two days, respectively, prior to operation, each of whom showed a good-sized pyloric tumor at operation. Thus, a single negative x-ray study should never be accepted as conclusive. If clinical signs persist and the diagnosis remains in doubt, roentgen examination

should be repeated one or more times at intervals of a few days. Pylorospasm, which may be most difficult to differentiate from stenosis clinically, is usually not associated with marked delay in gastric emptying by x-ray, and a 24-hour gastric residue is not found. Retention of 75% of a barium meal at the end of five or six hours and a large residue at the end of 24 hours may be accepted as definite evidence that pyloric obstruction exists.

One patient of this series showed not only marked pylorospasm but also marked cardiospasm by x-ray (Fig. 4). This picture may be confused with the chalasia of Berenberg and Neuhauser,[9] which was not encountered in this series.

Experience has demonstrated to us that in a large general hospital the care of these infants should be under the constant supervision of the pediatrician. Equally important, surgery of this type should be concentrated in the hands of a few if the mortality rate is to be kept low.

In this series, operation for pyloric stenosis was never considered a dire emergency, and adequate time was taken for hydration, restoration of electrolyte balance, and transfusions as needed. Particularly, any evidence of tetany was considered a strong indication for postponing surgery. On the other hand, an infant showing no relief of obstructive signs on intensive medical management of three to four days' duration was subjected to surgery without further delay, once adequate preoperative preparation had been instituted. Blood chemistries were rarely taken.

In the immediate preoperative period, gastric intubation with a No. 12 French urethral catheter was routinely performed, and the stomach was emptied of its contents. By this maneuver the danger of aspiration of vomitus during or immediately after anesthesia is obviated.

TECHNIQUE OF OPERATION

Our anesthesia of choice for these infants was open-drop ether. More recently cyclopropane has been used with good results. An upper abdominal right rectus muscle-splitting incision approximately 2 in. (5 cm.) long is routinely employed. If an unsuspected enlargement of the liver is encountered or the lesion is found to be elsewhere in the abdominal cavity, this type of incision may prove advantageous over the oblique or transverse type. Bleeding points are tied with 4-0 nonabsorbable surgical (silk) sutures or cotton. The distal segment of the stomach is grasped gently, and the antrum and pylorus are delivered into the wound. The pylorus is grasped between the thumb and index finger of the left hand and firmly compressed before incision into its musculature is performed (Fig. 5). Compression of the pyloric mass in this fashion is important, since thereby the operator is best able to judge the extent of the tumor, particularly with regard to its exact distal edge. If incision into the tumor is carried to a point about 2 mm. proximal to its distal edge, as estimated by palpation, or to the pyloric vein, if this be visible, perforation of the duodenum is not likely. The incision should not be extended deeply; rupture of the deeper musculature is accomplished by gently but forcibly spreading the pylorotomy wound apart by repeatedly opening the jaws of a small hemostat therein. The submucosa can be recognized by its shiny pearly-grey appearance (Fig. 6). After pylorotomy has been adequately performed, the submucosa along the entire course of the wound is free of constricting transverse muscle fibers and should pout up into the incision. Relief of pyloric obstruction should then be

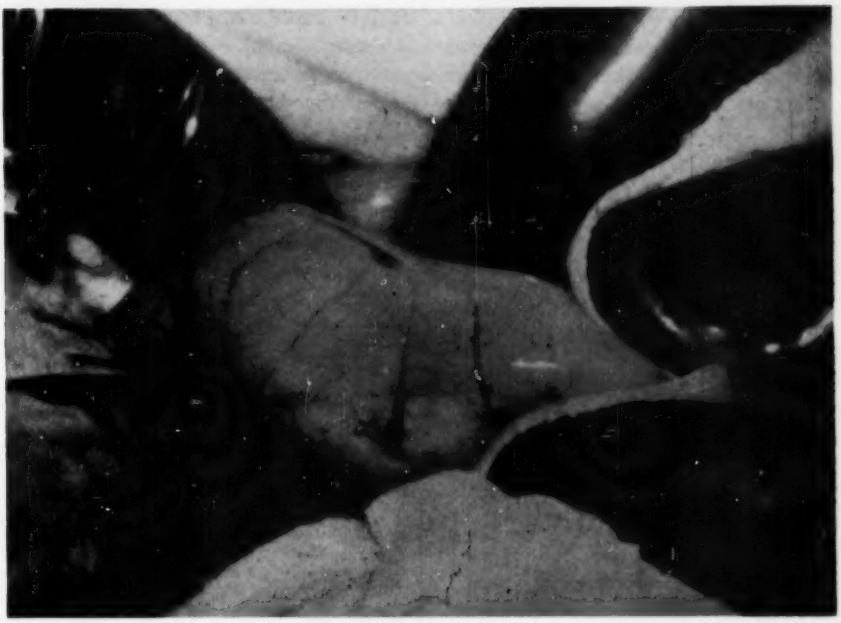

Fig. 5.—Pyloric tumor grasped between left thumb and forefinger of operator. Antrum of stomach is pulled to left by assistant. Pyloric vein is visible at distal end of tumor.

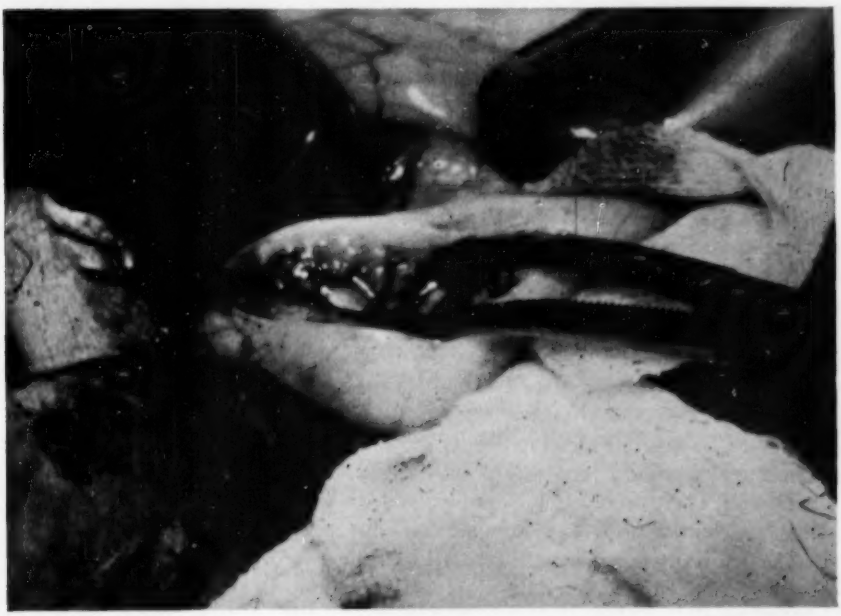

Fig. 6.—Small portion of shiny mucosa is visible in center of pyloromyotomy wound.

demonstrated by compression of the stomach antrum. This maneuver will also demonstrate any unsuspected perforation of the duodenum at the distal end of the pylorotomy wound. Should a perforation be present, its closure may be accomplished with a purse-string suture of 5-0 arterial silk, reinforced by omentum or a V-shaped flap of pyloric muscle. There was one perforation of the duodenum which was treated in this series. Slight oozing of blood from the incised pylorus is to be expected and is of no consequence; further bleeding is easily controlled by the temporary application of small piece of absorbable gelatin sponge. The abdominal wound is closed in layers with continuous 2-0 chromic atraumatic absorbable surgical (gut) sutures to the peritoneum and posterior fascia and interrupted 4-0 silk sutures to the fascia and skin. In all cases, stay sutures of No. 32 stainless-steel wire are carried through all layers down to the peritoneum at ¼ in. (0.6 cm.) intervals and tied over a small gauze roll. Closure of the peritoneum is best commenced at the lower end of the wound, since thereby the defect in the peritoneum last sutured is protected by underlying liver, and the danger of catching bowel or omentum in the last stitch is minimized.

In this series the postoperative care was carried out primarily by the members of the pediatric department. Fluid and caloric requirements were met by intravenous injections of 5% dextrose in water or saline, and we agree with Swenson [10] that in infants intravenous infusions are far preferable to subcutaneous administration of fluids. After recovery from anesthesia, the infant was fed sterile water, and if this was retained, he was started on a graduated feeding regimen of first barley water, then barley water and breast milk, and finally whole breast milk. If vomiting occurred, the regimen was recommenced from the beginning.

ANALYSIS OF RESULTS

Thirty-seven infants were operated upon with the preoperative diagnosis of pyloric stenosis, two more with a tentative diagnosis of pylorospasm, and two others with preoperative diagnoses of "intestinal obstruction" and "probable malrotation" respectively. Both of these latter patients were found to have congenital hypertrophic pyloric stenosis at operation. The two infants with a preoperative diagnosis of pylorospasm were found to have this condition. In each case the Ramstedt procedure was carried out. Of the 37 patients with preoperative diagnoses of pyloric stenosis, 34 were proved to have this condition at surgery. In the remaining three, the final diagnoses were, respectively, malrotation with duodenal compression, pyloric compression by a congenital band continuous with the round ligament of the liver, and duodenal constriction secondary to a cholecystoduodenal band with pyloric angulation. In the latter two cases it was impossible to determine whether a band or pylorospasm was the true obstructing agent. In each case, therefore, the band was divided, and a Ramstedt procedure was then performed.

Twelve, or one-third, of the infants showing pyloric stenosis at operation were females, a proportion in contrast to most series, wherein 80 to 90% of the patients are males.§ There were eight Negro children in this series, seven of whom were shown at operation to have pyloric stenosis; one, pylorospasm.

The age of these patients, in contrast with those of other series, was markedly variable. The youngest was 3 days old and the oldest, 4 months old. The duration

§ References 1, 2, 6, and 11.

of symptoms ranged from three days to five weeks. A tumor was unequivocally palpable by us in only 17 of the 36 who were found to have pyloric enlargement at operation. X-ray studies with barium were carried out in 23 cases without untoward results.

In those cases in which the diagnosis was clear cut a minimum of time was utilized in reestablishing fluid and electrolyte balance. Longer periods of study were found necessary when the diagnosis was in doubt. So long as medical management appeared to be sustaining these children with equivocal findings, operation was deferred. As a result, the preoperative period in the hospital varied from 1 to 33 days.

Pyloromyotomy was carried out in all cases, and, in addition, division of the obstructing ligaments, or bands, was done as indicated.

Postoperatively, all but one of these patients did well, the only relatively common untoward incident being occasional vomiting. This was of an important degree in only one case in which pyloromyotomy had been done for pylorospasm. In the one case in which perforation of the duodenum occurred at operation, Wangensteen suction through a No. 12 French nasal intragastric catheter was maintained for the first two postoperative days as a precautionary measure. This baby had an uneventful convalescence.

The average hospital stay following operation was of extraordinarily long duration in this series. This was in part because the patient was not discharged until all sutures had been removed (10 to 12 days) and until it could be demonstrated that the infant was gaining weight; in several instances the hospital stay was prolonged because of unsatisfactory conditions at home. The duration of this postoperative stay varied from 12 to 58 days, the average being 20 days. Other than vomiting, the only complications noted in this entire series were three cases of localized saphenous phlebitis at "cut down" sites and one case of rather severe diarrhea, which was controlled after three days.

COMMENT

The findings in this series indicate that, in contradistinction to the usual clinical teaching, pyloric stenosis may occur at any time in the neonatal period. Persistent vomiting in the newborn should be carefully studied with this possibility in mind. If central nervous system damage, obscure infections, or a faulty feeding regimen can be ruled out, and no pyloric tumor can be palpated, x-ray studies of the upper gastrointestinal tract should be performed. The finding of obstruction at the pylorus, or in the first portion of the duodenum, is sufficient justification for exploratory laparotomy, and this should be carried out forthwith. We do not think it justified to defer operation indefinitely until a "tumor" can be palpated. We feel that further advance in the treatment of this disease should now be directed at earlier diagnosis, since thereby the patient can be brought to surgery in better condition. In our experience, not infrequently x-rays will establish the diagnosis before the tumor can be felt; occasionally the reverse also obtains: The tumor may be palpated in the presence of a normal-appearing barium study.

In two of the cases herein reported, the operative findings were merely of pylorospasm, but pyloromyotomy done, nevertheless, as an investigative maneuver, gave good result in one and an equivocal result in the other. The phenomenon has

been commented on previously.[12] The satisfactory result in one of these two cases suggests that pylorotomy may be worth while in such instances when the failure to find any other cause for obstruction leads to the diagnosis of pylorospasm by exclusion and when there is complete refractoriness to medical management.

The right rectus muscle-splitting incision was used in all cases because it was felt to give better exposure and allowed more latitude for exploration.

Since the vertical incision has been criticized || because of the increased possibility of dehiscence, it is to be noted that there was no dehiscence or subsequent wound hernia in any case of this series.

Sixteen patients have been followed from three months to three years following surgery. All were doing well nutritionally.

CONCLUSIONS

The cases of congenital hypertrophic pyloric stenosis occurring in a large city hospital over a three-year period are reported.

The differential diagnostic problems which must be considered in establishing the diagnosis of congenital hypertrophic pyloric stenosis are discussed.

A description of the surgical technique and abdominal findings in this series is presented.

It is suggested that when no pathology can be found at operation, thus leading to a diagnosis of pylorospasm, the Ramstedt pyloromyotomy should nonetheless be performed.

There were no deaths in this series. The only complications were occasional vomiting and three cases of mild phlebitis from "cut downs."

A comparison of mortality rates in a large city hospital emphasizes the advisability of concentrating the pediatrics surgery in the hands of a few.

It is felt that the mortality rate is lowered by early surgical intervention in cases refractory to medical therapy, that early surgical intervention is preferable to temporizing for long periods in an effort to establish a positive diagnosis while the patient's condition deteriorates.

Drs. Eli Friedman, John Treanor, James Redmond, A. Nathan Caplan, and John Faherty gave permission to include their private cases in this report.

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PENETRATING PEPTIC ULCER WITH MASSIVE HEMORRHAGE AS AN INDICATION FOR EMERGENCY GASTRECTOMY

Method of Clinical Diagnosis

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THE FUNDAMENTAL problem in the management of massive peptic ulcer hemorrhage has been to choose which patient will respond to medical management and which patient will require emergency surgical treatment. At present the most popular method of management consists of initial medical treatment with blood replacement, followed by prompt surgery if there is a recurrence of significant hemorrhage. Stewart [1] has used the actual amount of hemorrhage itself as an indication for the necessity of surgical treatment. This of course eliminates the risk of a patient exsanguinating, but has the disadvantage of doing emergency gastrectomy for hemorrhage in a significant number of patients who would respond to adequate medical management. In our series of 150 cases of proved peptic ulcer with acute massive hemorrhage, 60 patients were successfully treated medically. The age of the patient with massive peptic ulcer hemorrhage has been used in the past [2] as the principal indication for medical or emergency surgical treatment. Although there is no question that atherosclerotic changes in the vascular system of patients in older age groups, with resultant inability of vessels to contract properly, play a part in recurrent or continued bleeding, it has also been proved by experience that significant numbers of patients in the age group below 40 can have intractable life-threatening hemorrhage requiring emergency surgical treatment. In our series of 84 patients with massive ulcer hemorrhage who required emergency surgery or who bled to death, there were 27 under 50 years of age and 16 under 40.

It has been our feeling that the most significant factor in determining whether or not a patient will respond to medical management lies in the pathology of the ulcer itself.[3] We have been particularly impressed, as have others,[4] with the significant role that the penetrating type of ulcer plays in intractable massive peptic ulcer hemorrhage.

PENETRATING ULCER AND HEMORRHAGE

While it is true that erosive and superficial ulcerative lesions can cause serious hemorrhage, it is the penetrating type of ulcer with erosion of major branches of the gastroduodenal and left gastric artery respectively which is completely

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unsuitable for any type of treatment other than direct surgical control (Fig. 1). When one visualizes these ulcers with their callous type of base and erosion of large arterial trunks, one cannot help but feel that they are no more suitable to medical management than would be a defect in a major arterial trunk of an extremity. In

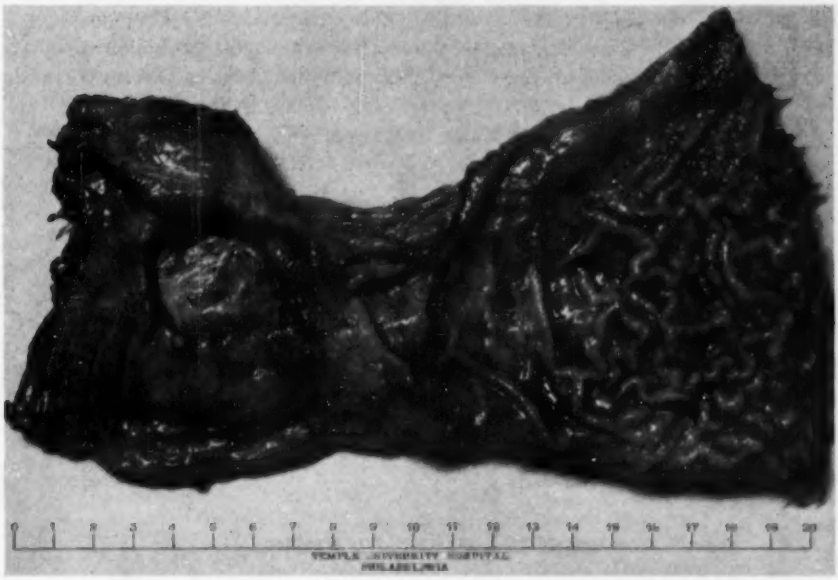

Fig. 1.—Postmortem specimen from patient exsanguinating because of a penetrating duodenal ulcer. Slit-like aperture in gastroduodenal artery can be seen in right upper portion of ulcer.

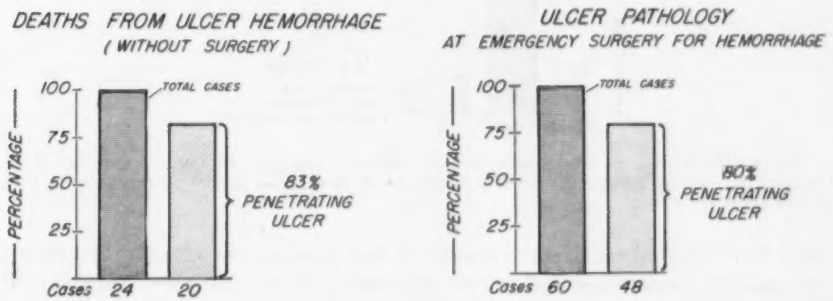

Fig. 2.—Approximately 80% of patients who either bled to death or required emergency surgery for acute massive ulcer hemorrhage had a penetrating type of duodenal or gastric ulcer.

our series of 150 patients with proved massive peptic ulcer hemorrhage, emergency surgery was necessary for bleeding or the patient bled to death in 84 cases. Of these 84 patients with intractable hemorrhage, a penetrating type of peptic ulcer was present in 68, or 80% (Fig. 2). The remaining 20% of patients in this group with intractable hemorrhage had ulcers of the more superficial type.

CLINICAL DIAGNOSIS OF PENETRATING ULCER

Because of the high incidence of penetrating ulcer in those patients with intractable hemorrhage, we became interested in means of establishing a diagnosis of this type of ulcer when the patient with massive hemorrhage is first seen. It is obvious that gastroscopy is of little value during the stage of acute bleeding and of no value in visualizing duodenal ulcers. Likewise, it is extremely difficult to diagnose a penetrating ulcer by x-ray examination unless there is a definite extraluminal pocket. An effort was then made to determine whether the diagnosis of penetrating ulcer

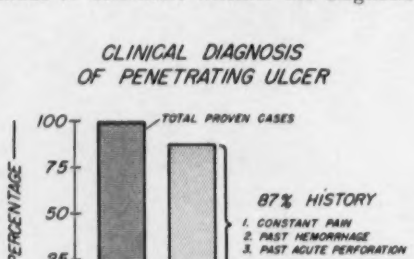

Fig. 3.—A correct clinical diagnosis of a penetrating type of ulcer as the source of the acute massive hemorrhage could have been made in 87% of proved cases on the basis of one or more of the above historical factors.

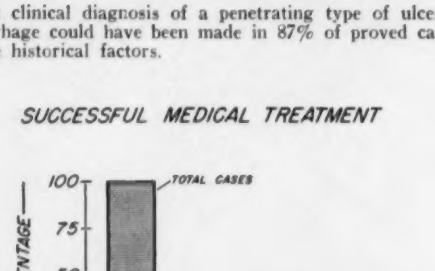

Fig. 4.—Only 18% of successfully treated medical cases showed one or more of these historical factors, as compared with 87% incidence of these same factors in the patients with proved penetrating ulcer.

could be established on clinical grounds. It was found in the group of 68 patients with massive hemorrhage and proved penetrating ulcers, that there were several factors in the history which were felt to be significant: (1) constant pain preceding the hemorrhage, (2) previous manifest hemorrhage, and (3) previous acute perforation. With use of any one or more of these factors as an indication of penetrating ulcer as the source of the hemorrhage, a correct diagnosis would have been made in 87% of those patients with proved penetrating ulcer as the source of massive hemorrhage (Fig. 3).

When one compares the 87% incidence of these historical factors in the group with proved penetrating ulcer with the 18% incidence of these same historical fac-

tors in the group who were treated successfully by medical means (Fig. 4), the implication is that there were few penetrating ulcers in the patients successfully treated medically.

SUMMARY

In our series of 150 cases the patient with massive peptic ulcer hemorrhage and one or more of the following historical factors (1) constant pain preceding the hemorrhage, (2) previous manifest hemorrhage, and (3) previous acute perforation, had an 87% chance of having a penetrating ulcer as the source of hemorrhage. These ulcers are pathologically unsuitable for medical treatment. Patients with these historical factors and massive hemorrhage should have immediate blood replacement and prompt surgical treatment.

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PEPTIC ESOPHAGITIS

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THE PATIENT with peptic esophagitis has long been neglected, allowing progression of the disease to chronic ulceration, fibrosis, and stricture. This neglect has been due to a failure of appreciation of the pathogenesis and clinical course of the disease and an ineffective therapeutic program.

Peptic esophagitis is an inflammatory process of the esophagus which occurs when there is a persistent association of gastric or intestinal secretions with the nonresistant squamous mucosa of the esophagus.[1] The reflux of these secretions into the esophagus is due to nonfunction or failure of the cardiac sphincter mechanism. This is usually caused by an associated sliding hiatus hernia, persistent vomiting, or the surgical excision or disruption of the cardiac sphincter (Table 1).

The major factors involved in the prevention of regurgitation into the esophagus by the cardiac sphincter can be identified. The inferior constrictor muscle of the esophagus and the oblique gastric muscles about the lower end of the esophagus produce a muscular sphincter by their purse-stringing action. In addition, the right diaphragmatic crura helps by maintaining the position of the stomach below the diaphragm and the acute angle at which the esophagus joins the stomach. A minor contribution to the competency of the cardiac valvular mechanisms is the pressure effect produced by air in the cardia, which tends to collapse the esophagus and increase the esophagocardiac angle.

Persistent vomiting occurring after surgery, during pregnancy, or from duodenal obstruction may overcome the normal cardiac sphincter and initiate esophageal inflammation. Operations which destroy or disrupt the cardiac sphincter mechanism frequently result in reflux esophagitis from regurgitation.

The majority of patients with peptic esophagitis have an associated sliding hiatus hernia. An understanding of the physiology of gastric reflux and activity in this condition is essential.

Aylwin [2] found that the enzyme activity of pepsin was responsible for the esophagitis, and its severity depends upon the factors which modify peptic activity and the presence of natural esophageal defenses. These factors include the diaphragmatic hiatus, vagus nerve function, esophageal and salivary secretions, presence of enough acid-producing glands within the hernia itself to activate the pepsin, and the competency of the cardiac sphincter above the hiatus hernia.

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The origin of vagal hyperactivity is perhaps constitutional, as the special liabilities of patients with hyperchlorhydria to develop peptic ulceration of the esophagus has been correlated with the high incidence of associated gastric and duodenal ulceration.*

The esophagitis itself once established may produce vagal stimulation which could be responsible for the increased acid-peptic gastric secretions. This tendency of esophagitis to establish a vicious cycle through vagal stimulation is readily apparent and accounts to some degree for the progressive nature of this disease.

Contrary to former beliefs,† it is our opinion, as well as that of others,‡ that the short esophagus with hiatus hernia is not congenital but usually an acquired condition due to peptic esophagitis which produces spasm, scarring, and permanent stricture of the esophagus.

Ectopic gastric mucosa occurring in the esophagus has been implicated as a possible cause of esophagitis; however, these ectopic foci are usually very small

TABLE 1.—*Etiology of Cardiac Sphincter Incompetence*

	Cases	
	No.	%
Hiatus hernia	98	76.0
Surgical excision or destruction of sphincter	17	13.0
Esophagogastrostomy for carcinoma	10	....
Esophagogastrostomy for lye stricture	3	....
Postcardioplasty	4	....
Persistent vomiting	15	11.0
Duodenal ulcer	13	....
Congenital duodenal stenosis	1	....
Epilepsy	1	....
Total	130	100

and unless some other mechanism is present to allow gastric reflux the normal protective salivary and esophageal secretions are adequate to neutralize this acid-pepsin production.

SYMPTOMS

Peptic esophagitis produces substernal pain, nausea, regurgitation, vomiting, dysphagia, aphagia, hematemesis, and melena. These symptoms occur characteristically either singularly or in various combinations according to the stage of the inflammatory process.

In acute esophagitis, pain is the predominant symptom and is manifest by a burning substernal sensation, sometimes described by the patient as "heartburn." The duration of pain at the onset is short and differs from gastric or duodenal ulcer pain in that it is usually relieved immediately by swallowing bland food or antacids. If the inflammatory process progresses, pain is no longer relieved by foods or antacids, and it may persist for two or three hours after eating. Patients become so fearful of swallowing that they refuse to eat, and marked weight loss and anemia are frequent findings. The pain is characteristically substernal in origin but may

* References 3 and 4.

† References 5 and 6.

‡ References 7 and 8.

spread upward into the jaw, ear, hard palate, or radiate through to the back, or down the arm simulating acute coronary insufficiency. The radiation of pain to these multiple and distant sites is explained on the basis of the sympathetic and parasympathetic innervation of the esophagus which is cervical, thoracic, and vagal in origin and is similar to the innervation of the heart. Many patients describing such a pattern of radiation are immediately labeled psychoneurotics and progression of the disease continues. Often patients will describe the sensation of epigastric fullness and dull pain upon leaning forward or upon lying down, especially after meals.

As the esophagitis progresses, dysphagia from muscle spasm and edema becomes the predominating symptom. Ulceration of the esophagus occurs with subsequent fibrosis, shortening, and circumferential cicatrization. Regurgitation of saliva, especially in the early morning before arising, is distressing, and the patient soon finds that he is able to take only a liquid diet, or he eats solid food and develops complete esophageal obstruction. Occult blood is practically always demonstrable in the stool, and severe hematemesis and melena occur in about 10% of the cases (Table 2).

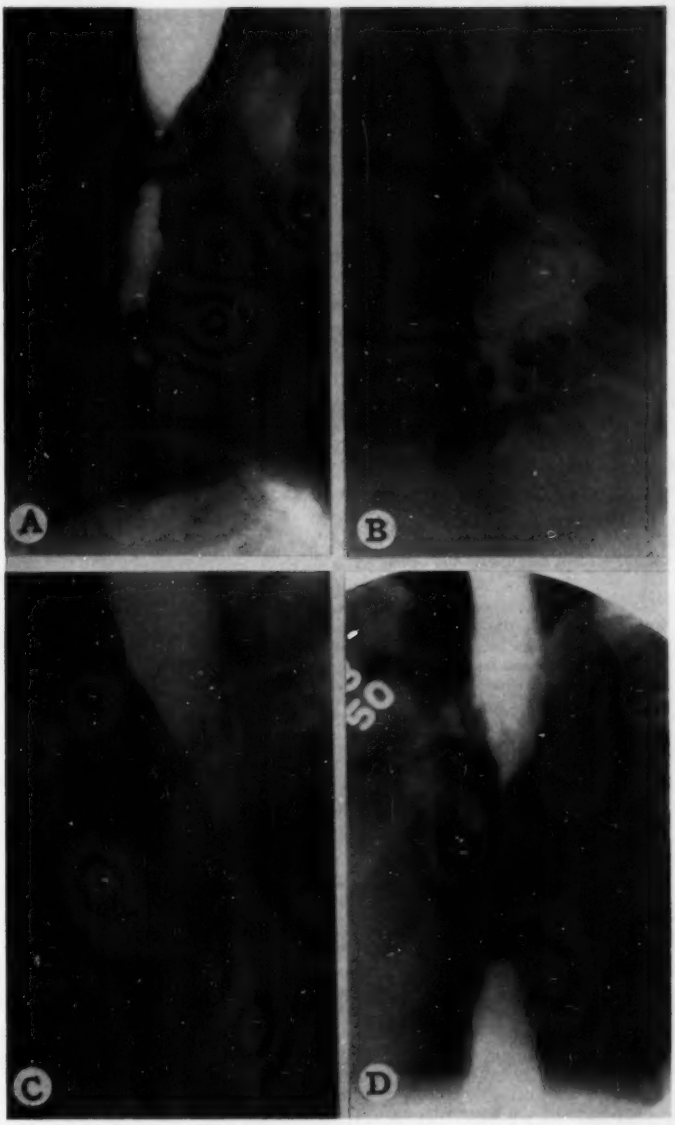
TABLE 2.—*Symptoms*

Symptoms	Cases	
	No.	%
Dysphagia	100	77
Substernal pain	91	70
Regurgitation	52	40
Weakness, anemia	44	34
Hemorrhage, hematemesis	13	10

Perforation of the esophagus is an occasional complication and developed in one of our patients. The development of carcinoma of the esophagus in the presence of peptic ulceration was not experienced in our series but has been reported by others.[9]

DIAGNOSIS

The diagnosis of peptic esophagitis is not difficult if one is aware of the symptom of substernal pain with its multiple points of radiation, regurgitation of saliva and food, and dysphagia. In early acute esophagitis, with or without ulceration, the barium swallow may be misleading, as it may show no abnormality except a slight delay of the passage of the emulsion through the lower esophagus. The esophageal ulcerations are demonstrated best with the patient in slight Trendelenburg position while swallowing the opaque medium. This maneuver will fill the esophagus below the area of spasm or constriction, frequently revealing an "hour glass" type defect or an ulcer crater (Fig. *A*). Coincidental hiatus hernia is so frequent that esophagitis should always be suspected when a hiatus hernia is demonstrated by x-ray examination (Fig. *B*). The esophagus above the point of constriction may be slightly dilated, but there is never the megaloesophagus so frequently seen in achalasia. The x-ray picture is not always so characteristic, however, and a long smooth constriction (Fig. *C*) or an irregular stricture resembling carcinoma may be present (Fig. *D*).



A, esophageal stricture with funnel-shaped or "hour glass" deformity due to peptic esophagitis. *B*, barium swallow performed with the patient in slight Trendelenburg position, demonstrating esophageal stricture with associated hiatus hernia. *C*, long smooth constriction produced by ascending type of esophagitis above the esophagocardiac junction. *D*, esophageal ulceration with almost complete obstruction. A diagnosis of carcinoma of the esophagus was made by the roentgenologist because of the ragged irregular proximal esophagus. Exploratory thoracotomy with resection of the lesion and esophagogastrostomy proved it to be stenosis due to ulceration of acid-pepsin origin.

Esophagoscopy establishes the diagnosis and should always be done to rule out carcinoma. In acute esophagitis the mucous membrane is firey red with edema and friability of the tissue, while the chronic ulcer is flat, covered with a whitish-yellow exudate and with mucosal congestion about its base.

The differential diagnosis is usually not difficult, but occasionally cholecystitis, coronary insufficiency, peptic ulcer of the stomach or duodenum, anxiety tension states, achalasia, and carcinoma of the esophagus may simulate esophagitis.

REPORT OF CASES

One hundred and thirty patients with peptic esophagitis are reported on in this series, covering a 15-year period from 1938 to 1953. There were 73 males and 57 females, and the average age was 51 years.

Hiatus hernia was found to be the cause of cardiac sphincter incompetence in 76%, surgical excision or destruction of the sphincter in 13%, and persistent

TABLE 3.—*Method of Treatment*

	Cases	
	No.	%
Group I		
Medical without esophageal dilatation	47	35
Group II		
Medical with oral dilatation	46	35.0
Repair of hiatus hernia and oral dilatation	15	11.6
Gastrostomy and retrograde dilatation	9	7.0
Subtotal gastric resection and retrograde dilatation	1	0.8
Subtotal gastric resection and repair of hiatus hernia	2	1.6
Phrenicoexeresis and oral dilatation	1	0.8
Group III		
Medical regimen after		
Resection of esophageal stenosis with esophagogastrostomy	7	5.3
Cardioesophagoplasty above stenosis	2	1.6
Total	130	

vomiting in 11% of the cases (Table 1). An associated duodenal ulcer was found to be present in 13 cases.

The most frequent complaint was dysphagia, which occurred in 77%; substernal pain was a close second with 70%; followed by regurgitation, 40%; weakness, 30%, and hemorrhage, 10% (Table 2). Nausea, vomiting, headache, recurrent melena, unexplained secondary anemia, and weight loss were among the other symptoms and signs.

Acute esophagitis without ulceration was found in 47 instances. This diagnosis was made primarily from history and from x-rays showing a delay in the passage of barium through the lower esophagus. Patients in this group refused esophagoscopy, and ulceration, if present, was not demonstrated by x-rays. Of 101 patients esophagoscoped, 82 were found to have evidence of acute or chronic ulceration. Roentgenological studies with barium revealed a "sliding type" hiatus hernia to be present in 98 cases and a duodenal ulcer associated with peptic esophagitis in 13 cases. In three cases there was a hiatus hernia and a duodenal ulcer in conjunction with a peptic ulcer of the esophagus.

For convenience in discussion of treatment, the 130 patients were divided into three groups according to the stage of the inflammatory process at the time of admission and the type of therapy instituted:

Group I: Acute esophagitis with or without demonstrable ulceration not requiring esophageal dilatation (47 patients).

Group II: Esophagitis with chronic ulceration and stricture requiring oral or retrograde dilatation (74 patients).

Group III: Chronic esophageal ulceration with stenosis (nine patients). The method of treatment employed is numerically shown in Table 3.

TREATMENT

Group I (Acute Peptic Esophagitis).—Elevation of the head of the patient's bed on 8 in. (20 cm.) blocks is essential in preventing gastric reflux and should be a permanent part of the therapeutic program even after all symptoms have subsided. This simple but effective maneuver takes advantages of the forces of gravity to preclude night-reflux.

Perhaps the commonest error in treating acute peptic esophagitis is the institution of multiple bland-diet feeding as used in the treatment of gastric and duodenal ulceration. Although the multiple feedings tend to neutralize the irritating gastric juices, they also stimulate gastric motility and the production of more hydrochloric acid and pepsin. The esophagus is at rest normally except during deglutition; therefore, in order to decrease the esophageal peristaltic activity three large liquid or semisolid feedings are advocated. A glass of water should follow each meal to clear the lower esophagus of food particles, and patients are given a liquid antacid which is taken at the slightest sign of substernal discomfort and at night before retiring. For patients who are overweight and have a hiatus hernia, a reduction diet is imperative. Atropine administered 30 minutes before meals will reduce spasm and gastric secretions.

The introduction of parasympatheticolytic blocking agents, such as methantheline (Banthine) bromide [10] which reduces gastric acidity and motility, has been an adjunct to the treatment of esophagitis. Antibiotics are of value in reducing secondary bacterial infection.

Treatment should be continued until the patient has had no pain or discomfort for at least two weeks, three consecutive stools have contained no occult blood, and x-rays show no ulcer crater or delay of barium passage through the lower third of the esophagus. The patient should follow a bland diet for an additional two or three months.

Group II (Esophagitis with Chronic Ulceration and Stricture).—Dysphagia is the usual complaint of patients with chronic ulceration, and the relief of this distressing symptom is based on the degree of stricture, the anatomical position of the esophagocardiac junction, and the associated gastric and duodenal disease. A conservative program of therapy is followed in these cases as long as the symptoms can be controlled, and the measures outlined for Group I patients are employed in conjunction with oral dilatation of the esophagus with bougies.

The patient swallows a string and bougies are passed, this string being used as a guide. Dilatations should never be done by blind bougienage, as the danger of esophageal perforation is considerable.[9] Seventy-four patients were treated by

dilatation (Table 3). Approximately one-fourth of this group required repeated oral dilatations, and at first the dilatations were performed at weekly intervals but subsequently it became necessary only at six-month intervals. The course of the inflammatory process is followed by barium x-rays and esophagoscopy.

Occasionally the degree of stricture is such that oral dilatation is hazardous or impossible. The endoscopist is usually forewarned by complaints of severe pain, and in such cases a gastrostomy is performed with retrograde dilatations. The gastrostomy is also advantageous in maintaining an adequate nutrition and placing the esophagus at rest. Nine patients had a gastrostomy with retrograde dilatation, and in six cases a satisfactory result was obtained and the gastrostomy could be closed in 12 months. Three of these patients required subsequent oral dilatation because of recurrent esophagitis.

Seventeen patients with sliding hiatus hernia and short esophagus due to peptic esophagitis were treated by repair of the hiatus hernia. These patients all failed to respond to conservative therapeutic measures, and the esophageal shortening was thought to be due primarily to esophageal spasm rather than inflammatory contracture. Five patients required postoperative oral dilatations of their preoperative esophageal stricture and have responded satisfactorily without recurrence of symptoms. Two patients developed a recurrence of their hernia, and two patients developed recurrent esophagitis which responded to medical therapy. Eight patients have remained completely free of symptoms. In nonobese patients who are discovered early before permanent esophageal shortening has occurred, repair of the hiatus hernia with restoration of the normal cardioesophageal mechanism is the treatment of choice. In repairing the sliding hiatus hernia, Allison [11] sutures the esophagophrenic ligament to the undersurface of the diaphragm, and the hiatal defect is repaired posteriorly, thus the normal anatomical relationships are restored. This method has been employed in our last seven patients without recurrence of symptoms to date.

Phrenicoexeresis with oral dilatation was used in one case of hiatus hernia with esophagitis, with relief of pain and aphagia. This patient refused hiatus hernia repair. His course has been unusually satisfactory; however, he has required oral dilatation at six-month intervals.

Gastric or duodenal ulcers are frequently associated with peptic esophagitis, and 13 such cases were found in this series. Three patients were treated by subtotal gastric resection. One was necessitated because of severe uncontrollable hemorrhage from a duodenal ulcer. Two patients had a subtotal gastric resection with repair of hiatus hernia because of the presence of esophageal ulcers, hiatus hernia, and duodenal ulcer with partial obstruction. Wangensteen [4] and Cornell and Winkelstein [12] have proposed the use of subtotal gastric resection for treatment of peptic ulceration of the esophagus, and their results were reported as being good after postoperative oral dilatation of the preoperative esophageal stricture.

Group III (Chronic Esophageal Ulceration with Stenosis).—The final stage of this disease process is cicatricial stenosis of the esophagus, and the surgical treatment consists of resection of the stenotic portion of the esophagus with esophagogastrostomy or esophagojejunostomy. Early in our series the stenotic lesion of the esophagus was bypassed by performing a cardioesophagostomy above the lesion in two patients. Their postoperative follow-up revealed an occasional recurrence of symptoms which responded to conservative measures. Clark and Adams [13]

reported on a series of five patients treated in this manner with comparatively good results. This method of treatment was abandoned, however, because it did not remove the primary lesion which was subject to perforation, hemorrhage, and development of carcinoma at the site of the stricture. Three cases of carcinoma developing in such a stricture were described by Benedict.[9]

Seven patients were treated by resection of the stenotic portion of the lower esophagus and intrathoracic esophagogastrostomy, with one postoperative mortality. Three patients have been asymptomatic; two have required subsequent medical therapy, and one has required hospitalization for oral dilatation. The physiologic mechanism whereby gastric juices reflux into the esophagus after excision of the cardiac sphincter is explained to the patients, and the necessity of sleeping with the head of the bed permanently elevated, antacid therapy, and prompt medical attention on recurrence of symptoms is impressed upon these patients postoperatively. All of these patients have regained their preoperative weight, and none have developed postcibal epigastric fullness.

In order to determine the incidence of esophagitis following esophagojejunal anastomosis, case histories of 12 patients who have had total gastrectomies performed were reviewed. Three patients developed esophagitis from reflux pancreatic and intestinal juices; two of these patients had a loop jejunal anastomosis with enteroenterostomy, and one had a Roux-Y type anastomosis. This is an incidence of 25% and compares with the studies of Scott and Longmire[10] in which they found 33% of their total gastrectomy patients with symptoms of esophagitis.

A comparative study was made of 100 cases in which the cardiac sphincter was excised and an esophagogastrostomy was performed at Duke Hospital, usually for carcinoma of the cardia. The incidence of postoperative esophagitis was 13%; however, this small incidence of esophagitis must be qualified for both series, as they represent short postoperative follow-ups due to early death from carcinoma for which the procedure was originally done.

In order to avert the symptoms of postcibal distress, loss of weight, and anemia associated with nutritional disturbances that accompany loss of gastric function in esophagojejunal anastomosis, we have preferred esophagogastric anastomosis in the treatment of stenotic esophagitis due to peptic digestion. In our experience, the incidences of esophagitis following loop esophagojejunostomy has been comparable with that following esophagogastrostomy, and the morbidity is much greater following the esophagojejunal type anastomosis.

COMMENT

It is apparent from the foregoing case presentations that the majority of patients with peptic esophagitis will have an associated hiatus hernia. Success in preventing stenosing esophagitis depends upon the early diagnosis of peptic esophagitis and repair of the hiatus hernias before esophageal shortening occurs.

The attitude of physicians toward patients with complaints of substernal pain radiating into the neck, jaw, shoulder, or arms has intrigued us. In reviewing the many charts of patients in this series the final diagnostic impression frequently has read as follows: "There is a hiatus hernia present but it is felt that the symptoms in this patient are entirely functional." All too frequently these patients return two or three years later with dysphagia, esophageal shortening, and stenosis—too late for hiatus hernia repair.

Three of 13 patients who had a duodenal ulcer associated with peptic esophageal ulceration developed esophageal stenosis requiring esophageal resection. The esophagitis developed in each case probably as a result of gastric reflux from duodenal obstruction. The fulminating course of the esophageal lesion is explained by the high acid-pepsin concentration of the regurgitated or vomited gastric juice. The high incidence of associated gastric and duodenal ulceration with peptic esophagitis has been reported by others.§

Three patients had esophageal ulcer, hiatus hernia, and a duodenal ulcer. Two were treated by subtotal gastric resection and hiatus hernia repair; the third patient, a poor operative risk, was not subjected to the combined operation, as the gastric resection was performed because of massive hemorrhage from a bleeding duodenal ulcer. One patient has been followed for five years, without recurrent esophagitis; the other patients are doing well postoperatively, but their postoperative course has been of too short a duration to draw conclusions. Subtotal gastric resection with repair of hiatus hernia in this particular type of patient is proposed because of the malignant nature of esophagitis when associated with obstructive duodenal ulceration. Benedict and Sweet [14] recognized this triad and reported six cases. Three were treated by oral bougienage and three with resection of the lower esophagus. One of the latter patients developed a postoperative stricture. This triad should be suspected in all cases of esophageal ulceration, and an effort should always be made to rule out an associated duodenal ulcer, especially when there has been a long history of vague epigastric discomfort. Demonstration of esophageal ulceration with benign stricture is not enough; an associated hiatus hernia and/or duodenal ulcer must be ruled out in order that definitive therapy can be instituted.

Most cases of peptic esophagitis can be controlled by conservative measures if the patient is cooperative and intelligent enough to understand the mechanics involved in its production and the value of continuous prophylaxis once the acute esophageal process has subsided.

Oral dilatation by string bougienage has proved to be a valuable adjunct to the nonoperative treatment, often relieving strictures which by x-rays and esophagoscopy appeared to be permanently narrowed.

In children and in the aged poor-risk surgical patient, gastrostomy with retrograde dilatation has been used with success when oral dilatation failed or was considered too hazardous. In nine patients this method was used, and in six the gastrostomy has been closed and these patients are now able to follow normal diets, although some require oral dilatations at six-month intervals.

When esophageal stenosis that cannot be relieved by dilatation occurs, esophageal resection with esophagogastrostomy is the procedure of choice, though Allison [15] advocates an esophagojejunal anastomosis. The preservation of gastric function of digestion has seemed more important than the minor episodes of recurrent esophagitis that occur after esophagogastrectomy, most of which can be prevented by prophylactic treatment.

SUMMARY

Peptic esophagitis is a serious progressive disease occurring most frequently with an associated sliding hiatus hernia. It is a grave complication following operative procedures which destroy or disrupt the cardiac sphincter mechanism.

§ References 3 and 4.

The pathogenesis and clinical course of the disease is discussed and an effective therapeutic program outlined for each stage of the inflammatory process. The value of esophagoscopy in achieving a correct diagnosis is emphasized.

Most cases of peptic esophagitis can be controlled by medical measures; however, patients must continue prophylactic treatment to prevent recurrence with esophageal shortening and, ultimately, stenosis.

Early repair of sliding hiatus hernia is indicated in the presence of esophagitis which fails to respond completely to medical management.

Subtotal gastric resection with repair of hiatus hernia is suggested for the treatment of patients with the triad of esophageal ulcer, hiatus hernia, and duodenal ulcer.

In peptic esophageal stenosis when all other therapeutic measures fail esophagogastrostomy is preferred to esophagojejunostomy because the over-all postoperative morbidity is lower. When esophagogastrectomy becomes necessary, then gastroenterostomy and vagotomy or subtotal gastric resection should be performed to protect the esophagus.

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SPECIAL PROBLEMS OF ACUTE APPENDICITIS IN MIDDLE AND LATE LIFE

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THE TOTAL picture of acute appendicitis at the present time has a number of components. Some of them are creditable. Others are not. The situation may be summarized as follows:

1. To our professional shame, this disease is still slaying its thousands. The death rate per 100,000 in the United States, which was 9.7 in 1900 and 15.2 in 1925, has fallen progressively since the latter date and last year declined to an all-time low of 1.7.[1] This remarkable decrease is somewhat less gratifying than on first glance it might seem when it is recollected that a death rate of 1.7 per 100,000 means that at least 2,600 persons died in this country in 1952 of a disease that has been well described as eminently treatable.

2. The mortality in simple acute appendicitis has always been low and is now minimal. Deaths in this type of disease are practically always caused by unforeseen events, which might as easily occur after any other abdominal surgery under anesthesia.

3. The mortality in acute appendicitis is now almost entirely concentrated in advanced disease, that is, acute appendicitis complicated by gangrene, rupture, peritonitis, and abscess formation. Although these complications remain potentially fatal, the outlook in all of them has been greatly improved by correct preoperative and postoperative management, including the adjunct use of antibiotic therapy.

4. There is a shocking misunderstanding of the proper use of antibiotic therapy in acute appendicitis. Antibiotics do not prevent the disease. It can occur, and can become complicated, and can prove fatal while patients are under treatment with antibiotics for other conditions. Nor are antibiotics a substitute for surgery in acute appendicitis. That concept of their use is as hazardous as it is baseless. The proper management of acute appendicitis remains prompt removal of the appendix. When that course is followed, antibiotic therapy is not needed at all. When the antibiotics are employed as a substitute for surgery, the patient is subjected to a gamble which does not always pay off. When it does, spontaneous resolution would probably have occurred without any treatment at all.

5. The mortality of acute appendicitis has always been disproportionately high in middle and late life. It is therefore surprising, in view of the massive literature growing up in the field of geriatrics, that only occasional articles are appearing concerning the disease in older persons.

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STATISTICAL DATA

The general facts of acute appendicitis in middle and late life are fully evident in the 7,613 surgical cases of the disease analyzed in seven separate studies from Charity Hospital of Louisiana at New Orleans, for the almost 20-year period ending in 1949. In the total series, as well as in each separate analysis, the age group over 39 years has accounted for the smallest proportion of cases and the largest proportion of deaths (Chart 1). The cases which occurred between 1950 and 1952 have not yet been analyzed in detail, and the hospital figures are employed with some hesitation for the reason that a good many of the cases filed as such are probably not acute appendicitis according to the rather strict criteria employed in previous analyses. The data are accurate enough, however, to show that the disproportions evident in earlier series are again evident in these cases.

Patients in the older age group, as would be expected in a progressively aging population, have made up an increasingly larger percentage of the total number of

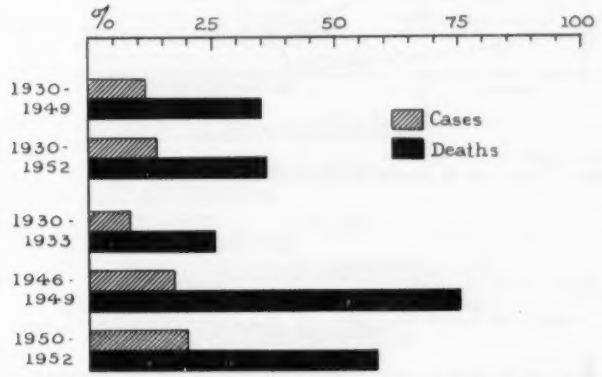

Chart 1.—Proportionate distribution of 1,228 cases and of 126 deaths in patients more than 39 years of age in a total of 8,883 cases and 352 fatal cases of acute appendicitis at Charity Hospital of Louisiana at New Orleans, 1930 to 1952.

cases as time has passed. In the first analysis, made in 1933, they constituted 8% of the total number. For the three-year period ending in 1952 they made up 19.5% of the total number. There seems no reason to doubt that the problem of acute appendicitis at the upper extreme of life will continue to become numerically greater with each passing year.

The case fatality rate, which has been studied according to age in each of these analyses, has always been much higher in the older age group (Chart 2). It was 12.14% for the 980 cases which occurred at Charity Hospital between 1930 and 1949, inclusive. This was more than twice as high as the rate in children through 12 years of age, and almost five times higher than the rate in the 13 to 39-year group. According to the hospital figures, the rate in the older age group was 2.9% for the 1950 to 1952 period, which is considerably lower than any rate previously accomplished. During these three years, however, the rate in children was only 1.19% and the rate in the 13 to 39-year group was only 0.2%.

The observations at the New Orleans Charity Hospital are borne out by Bureau of Census figures for 1952,[1] estimated on a 10% sample of deaths (Chart 3). It is startling to observe, in view of the infrequency of acute appendicitis at the upper extreme of life, that the largest number of deaths, 460, occurred in the decade end-

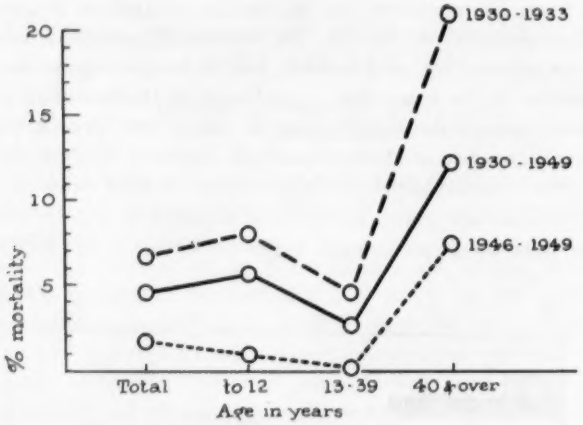

Chart 2.—Case fatality rate in relation to age of patients in 7,613 cases of acute appendicitis at Charity Hospital of Louisiana at New Orleans, 1930 to 1949.

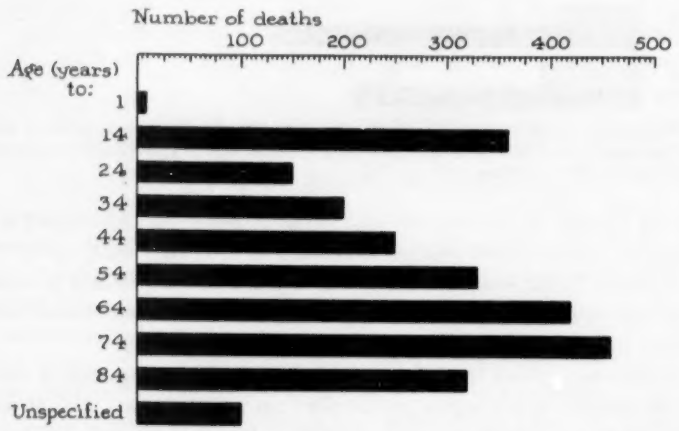

Chart 3.—Distribution according to age of 2,600 patients who died of acute appendicitis in the United States in 1952 (estimated on a 10% sample).

ing at 74 years and that the next largest number, 420, occurred in the preceding decade. Moreover, considerably more than half of the 2,500 deaths in which the age was stated occurred after 54 years of age. These numbers are likely to increase as more and more persons live into the upper age brackets.

PATHOLOGIC PROCESS

A good many observers, of whom I am one, explain part of the high mortality of acute appendicitis in older persons by the concept that the disease is inherently more serious at this period of life. This is not an unreasonable point of view. In addition to the vascular component more often present in older patients, the structure of the organ is altered.[2] Most lymphoid tissue has disappeared. Fibrous atrophic changes impair the circulation and make it impossible to meet the demands of the inflammatory process for an increase in the blood supply. The devitalized organ, especially when obstruction is a factor, presents little resistance to infection, and perforation soon occurs. The omentum and peritoneum often lack their pristine ability to localize infection, and general peritonitis results. Mixed infections, which are thought to progress more rapidly than monobacterial infections, are of frequent occurrence.

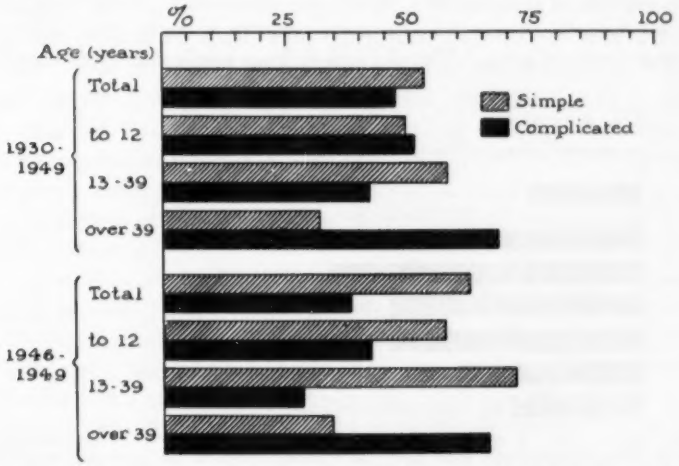

Chart 4.—Proportionate distribution according to age of 7,613 patients with simple and complicated acute appendicitis at Charity Hospital of Louisiana at New Orleans, 1930 to 1949.

Whether or not one regards acute appendicitis in aging persons as a special disease (*l'appendicite du vieillard, appendicite chez les malades agés* [3]), there can be no argument about the speed and seriousness of the pathologic process at this time of life. In the last series studied in detail from Charity Hospital, there were 22 instances of gangrene, 18 of rupture, and 1 of abscess formation in the 298 patients admitted within 12 hours of the onset of illness. Fifteen of these 41 complications occurred in the 28 patients over 39 years of age, who made up only 9.4% of the total number admitted during this period.

The Charity Hospital figures (Chart 4) also furnish further evidence of the seriousness of the pathologic process. The percentage of complicated cases in the total series was high, and the percentage in the most recent series analyzed was disturbingly high. What is most disturbing, however, is the fact that while there had been some improvement in this respect in the other age groups there was no improvement evident in the older patients. In the total series the proportion of advanced cases at this time of life was 68%. In the last series analyzed in detail it was substantially the same, 65.9%. These observations are in agreement with those

made by Wolff and Hindman.[4] In their series from the Cornell Division, Bellevue, and the New York Hospital gross perforation was twice as frequent in patients more than 60 years of age as it was in those in the decade ending at 40 years.

ATYPICAL MANIFESTATIONS

If we fastened our attention on one simple fact, the mortality of acute appendicitis in the older age group would promptly improve. This is a disease which is atypical in its manifestations in a large number of cases at any period of life. That fact or, more correctly, failure to recollect that fact still accounts for some of the mortality and for a major portion of the morbidity at all periods of life, when the clinical picture is such as to cause delay on the part of the patient and confusion and indecision on the part of the physician, even the physician who recognizes and appreciates the vagaries of this disease. Wolff and Hindman's [4] study bears out these statements. Atypical disease was far more frequent in their older patients than in the younger control group. The physical findings conformed to no consis-

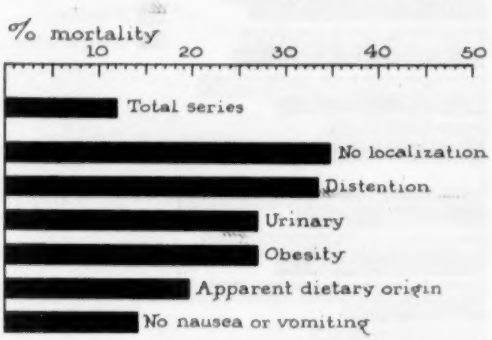

Chart 5.—Case fatality rate in relation to atypical symptoms and signs in 980 patients more than 39 years of age with acute appendicitis at Charity Hospital of Louisiana at New Orleans, 1930 to 1949.

tent pattern, and the range of variation in temperature and leucocytosis showed no consistent trend. Advanced disease was frequently associated with such a paucity of symptoms and signs that its true severity was not realized.

It has been well said that older patients with acute appendicitis enter the state of ill health slowly. In the Charity Hospital cases, symptoms often began in advance of the acute attack, sometimes considerably in advance, with a period of vague digestive distress. Pain was practically always present, but it appeared in all areas of the abdomen, including the left side. Often, both initially and throughout the attack, it was little more than discomfort. Other initial complaints included headache, backache, malaise, hiccoughs, and rectal bleeding. Such symptoms seem totally unrelated to acute appendicitis, and perhaps they are, but the argument is academic. They occurred in association with the disease; they confused the clinical picture, and the end-result was the same, delay in both diagnosis and treatment.

Nausea was frequent and inclined to be persistent. Vomiting was variable. In 108 cases, 11% of the total number, nausea, vomiting, or both symptoms were absent; 15 of these patients died, 13.9% (Chart 5). There were 4 deaths in the 31 cases in which diarrhea was present.

Fever showed the wide range typical of acute appendicitis at all ages but, generally speaking, was lower than in young persons, for the reason that the systemic response is more sluggish later in life. In almost a third of all cases, representing all varieties of pathologic process, the temperature did not exceed 99 F. In well over half of all cases, probably because of the same sluggish response, the pulse rate did not exceed 90. The white blood cell count tended to be lower than at other periods of life, but generalizations are not warranted. It ranged from 3,775 to 33,000 per cubic millimeter, while the polymorphonuclear leucocyte count ranged from 41 to 98%.

Physical findings were notably scanty. A uniform soft distention was common and apparently highly misleading (Chart 5). There were 30 deaths in the 89 cases in which it occurred. The process of localization took days, in contrast to hours in younger persons, and did not occur at all in 41 cases, 15 of which were fatal. It also did not occur at all in 12 of the 42 nonsurgical deaths in this age group over the 1930 to 1949 period. The reason that operation was not done in most of these cases was that the disease was not recognized ante mortem.

Rigidity was frequently absent because of muscular flaccidity, typical of the aging process, or was not evident because of obesity. Obesity is naturally not a physical finding peculiar to acute appendicitis, but it is frequent in older persons, and it complicates the diagnosis, as shown by the 13 deaths in the 35 cases in which it was present.

A number of circumstances confused the diagnosis in these cases. Overindulgence in alcohol and dietary indiscretions were among them (Chart 5). As outstanding instances, one Negro man became ill shortly after a heavy meal of pork chops, turnip greens, gin, and port wine, after which, for no apparent reason except, perhaps, some vague idea of prophylaxis, he took a dose of salts, thus adding the confusion caused by the purgative to the already confused dietary history. Another Negro man became ill after a midnight meal of pork chops, a whole coconut pie, and a bottle of beer. Both men were operated on within 24 hours; both had perforated appendices, and both died.

Another circumstance which confuses the clinical picture of acute appendicitis in later life is the presence of another organic disease. One death at Charity Hospital occurred in a patient who had been hospitalized for years for pulmonary tuberculosis. Another ocurred in a patient hospitalized for a long period of time with decompensated heart disease. In both instances all the attention was centered on the chronic ailments, and acute appendicitis was not even suspected until it was revealed at autopsy. In another instance the patient was thought to have nephritis, which he did, and an incarcerated inguinal hernia, which he had had several times in the past. In a number of cases acute appendicitis developed while the patients were hospitalized for other ailments, some trivial, some serious. In many of these cases the delay in diagnosis furnished further proof of the aphorism that the most unsafe place to have an acute illness is in the hospital, while undergoing treatment for a nonacute disease. It also brings to mind deTarnowsky's [5] warning that when acute appendicitis is suspected in older patients it may be imprudent to pay too much attention to a previous history of organic disease; his own patient's supposed recurrent coronary thrombosis proved to be acute appendicitis.

In some of the older patients at Charity Hospital the dangerous period of calm lasted for days instead of the hours it is likely to last in younger persons. In fact, the general mildness of the disease is evident in the number of patients who were refused admission to the hospital when they first applied, on the ground that they were not acutely ill, or who left the hospital against advice because they thought that they had recovered, or who were discharged by the staff in the same belief. Many of them returned to die.

DIAGNOSTIC CONSIDERATIONS

The history and physical findings in these older patients were frequently suggestive of almost any disease except acute appendicitis. The diagnostic possibilities listed after the admission surveys at Charity Hospital included gastroenteritis, cholecystitis, peptic ulcer with and without rupture, diverticulitis, Meckel's diverticulum, mesenteric thrombosis, tuberculous peritonitis, liver abscess, pelvic inflammatory disease, ovarian cyst with twisted pedicle, various types of cardiac and renal disease, amebiasis, pneumonia, malaria, hypoglycemia, and carcinoma of the stomach, rectum, and colon. One patient was being elaborately prepared for surgery for carcinoma of the cecum when he died. Another patient was operated on for a strangulated inguinal hernia, which he actually had, without discovery of the additional, entirely independent, acute disease of the appendix.

The list of diagnostic possibilities seems almost incredible when it is brought together, but there are few diseases mentioned for which there did not seem some justification. Malaria, hypoglycemia, and a number of other conditions were promptly eliminated early in the diagnostic routine, but the mere fact that a somewhat leisurely diagnostic routine was undertaken is another indication of the mildness of the symptoms and signs in older persons.

All patients with acute appendicitis are in a sense fortunate when they have the obstructive type of disease, or, rather, when their physicians recognize that they have it; all do not, although it is almost 50 years since it was first described. The older person is even more fortunate when he presents this type of disease. The onset is less severe, as a rule, than it is in younger persons, but it is still sufficiently violent not to be ignored by either patient or physician. In a number of instances in this series it was recognized that the patients had appendicitis, but the disease was considered chronic, subacute, or subsiding. The error was often disastrous.

The diagnosis was missed altogether in about 15% of the surgical cases at Charity Hospital, but, because some other acute abdominal state was suspected, operation was usually performed, though sometimes after considerable, and sometimes after fatal, delay. In Wolff and Hindman's [4] study the diagnostic error in the older group was 25%, against an error of 5.5% in the younger group. In the Charity Hospital series the mortality when operation was mistakenly or deliberately delayed was always far higher than in the younger group in corresponding circumstances.

The only solution of the diagnostic problem in acute appendicitis in older persons seems to be, as Wolff and Hindman [4] say, the adoption of a new set of diagnostic standards for this period of life. The most useful diagnostic aid is certainly the recollection of the possibility. The physician should bear this disease in mind whenever he encounters a patient with chronic or acute complaints associated with

the digestive process or with abdominal pain. In particular, he should not be misled by the mildness of complaints, which in the early stages, and frequently in the later stages as well, furnish little evidence of the mischief that may be afoot.[2]

Interesting proof of the diagnostic difficulties of acute appendicitis in later life is furnished by Hunter's [6] analysis of the diagnostic error in the case records of the Massachusetts General Hospital over the past 25 years. The 470 errors in the 2,600 cases were made in 100 different diseases, 11 of which were misdiagnosed 10 or more times. Eleven of the diagnostic errors concerned acute appendicitis. I have not looked up all of these cases, but I think it typical that the two which I came upon by accident, in 1948 [7] and in 1952,[8] were both in elderly persons, a 73-year-old man and a 65-year-old woman. It is also typical that in one presentation acute appendicitis was not mentioned at all in the course of the diagnostic discussion, while in the other the disease was mentioned only once, when it was stated that the condition for which the diagnosis was being sought could not possibly be acute appendicitis.

MANAGEMENT

The management of acute appendicitis in aging persons needs no extended discussion. It is just what it is in younger persons, prompt appendectomy. There is not the slightest reason for refusing operation to a patient advanced in years if the mere act of operation would not be fatal. There is far less reason for using conservative treatment at this age than at any other period of life.[9] Older persons do not tolerate toxemia well, and there is even less certainty than at other times of life as to what is occurring within the peritoneal cavity. Even the most ardent exponents of conservative therapy in advanced acute appendicitis do not consider it suitable late in life.

The type of anesthetic employed makes no difference, but inhalation anesthesia must not be profound. The mortality is not affected by the use of any particular incision. In the Charity Hospital series the McBurney incision and, more recently, the Davis and Rockey incisions, were widely used when the diagnosis was certain. Other incisions were more likely to be employed when it was not. The mortality with the latter types of incisions was slightly higher in younger persons and notably higher in older persons than with the McBurney or the Davis or Rockey incisions, but the poor results are more fairly attributed to delay and to the advanced stage of the disease than to the incision employed.

Still another reason for haste in the diagnosis and management of acute appendicitis in older patients is the diminished physiologic reserve at this period of life. Aging patients have a definite limit of tolerance. Nonetheless, postponement of operation for several hours to permit preoperative preparation is far wiser than immediate surgery on an ill, dehydrated, toxic patient, who possibly is also suffering from an organic disease.

Postoperative management is the same as at other periods of life, though certain precautions are necessary. The position should be changed frequently, especially immediately after operation, and the patient should be got out of bed as promptly as possible. Morphine is given only if it is clearly needed and is withdrawn as promptly as possible when it must be used. Replacement therapy is essential but must be carried out with great care because older persons frequently have a diminished cardiac reserve and are prone to develop pulmonary edema. Until oral nutrition can

be resumed, which should be as promptly as possible, fluids are given slowly by vein, in small amounts, with limitation of salt unless there has been an excessive loss of chlorides. The presence of an organic disease requires consultation with an internist. Death in acute appendicitis in aging persons is frequently the result of chronic organic disease, but that does not lessen the surgeon's responsibility for the fatality.

SUMMARY AND CONCLUSIONS

At the present time the mortality of acute appendicitis is almost entirely concentrated in advanced disease, and most of the deaths are occurring in persons over 39 years of age.

Whether or not the concept is accepted that the disease at this period of life is a special entity, there is no doubt of the speed of the pathologic process and the gravity of the prognosis.

The disease at this period of life is atypical in a large proportion of cases, and the manifestations are frequently deceptively mild. Diagnosis is correspondingly difficult, and therapy is delayed.

If those facts were borne in mind, there seems little doubt that both the mortality and the morbidity of acute appendicitis at this period of life would show a prompt and decided improvement.

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USE OF HYALURONIDASE IN SOFT TISSUE INJURY AND ITS INFLUENCE ON EXPERIMENTAL BONE REPAIR

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IN DISORDERS of the bones and joints, as in all other special fields of medicine, many primary conditions exist for which there is no definitive treatment, and also many troublesome complications are encountered that add to the difficulties of operation. During a clinical evaluation of hyaluronidase over the past three years, it became apparent that the enzyme might prove useful not only for rapid elimination of the extracellular fluid accumulations (edema, hematoma, hemarthrosis) that frequently complicate orthopedic conditions and delay operative treatment, but also for specific therapy or prophylaxis in certain surgical problems for which no satisfactory measures heretofore have been available.

The value of hyaluronidase as a useful adjunct in the treatment of fractures and other bone injuries has been stressed in an earlier report.[1] It seems apparent that hyaluronidase will find even greater usefulness through its ability to reduce soft-tissue swelling particularly as it relates to the problems of soft-tissue trauma and hemorrhage, impending Volkmann's ischemic contracture, and acute hemophilic hemarthroses and hematomata.

PHYSIOLOGY

In 1928 Duran-Reynals * reported the observation of a specific spreading factor in extracts of rodent testes; Meyer and his co-workers isolated a spreading factor from an extract of bull testis. The substance was named hyaluronidase.[4] Later investigations by Seifter and his associates † supplied further information on the physiologic and pharmaceutical action of this agent.

Hyaluronidase acts directly on the ground substance of normal connective tissue and on joint fluid by depolymerization of the mucopolysaccharide, hyaluronic acid, which forms the bulk of the intercellular substrate and is a constituent of synovial fluid. Breakdown of these natural barriers permits freer flow of the intercellular fluid, with more rapid dispersion and consequent absorption of extracellular fluid collections.

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Read in part before the Forum on Fundamental Surgical Problems, 38th Clinical Congress of the American College of Surgeons, New York, Sept. 24, 1952.

* References 2 and 3.

† References 5 to 9.

The enzyme itself has no propulsive properties but causes rapid spreading if local interstitial pressure is mechanically increased (externally by application of an elastic bandage or internally by the volume of the injected solution). The rate of diffusion is proportionate to the amount of the enzyme, and the extent is proportionate to the volume of the fluid injected.‡ The dermal barrier is partly restored in 24 hours and completely restored in 48 hours.[13]

The enzyme is available commercially as a vacuum-dried powdered extract of bull testis. It is supplied in vials containing 150 and 1,500 turbidity-reducing units § (TRU). Other manufacturers express hyaluronidase potency in viscosity-reducing units (VRU). One turbidity reducing unit equals approximately 3.3 VRU.

In clinical use, as described in this report, it has been our usual practice to dissolve 1,500 TRU of the powdered enzyme in 3 or 5 cc. of 1% procaine hydrochloride, thus giving 300 TRU or 500 TRU to each cubic centimeter of solution.

EXPERIMENTAL PROCEDURES

Since hyaluronidase disperses hemorrhage and hematoma in bone fracture, a question was raised as to whether repair might be inhibited and nonunion or delayed healing result. Before this enzyme could be used clinically with impunity, it was necessary to examine experimentally its specific affect on bone healing.

The method described by Bush and Garber was chosen.[14] Sixteen large chinchilla rabbits were used. With the animal under pentobarbital sodium anesthesia, a constant sized bone disc was removed from the iliac crest with a ¾ in. (1.9 cm.) tubular saw fashioned from a cork drill so that the bed and the graft were always of the same shape and size. The graft was stripped of periosteum and replaced and held in position by means of tantalum wire which was threaded through drill holes in the graft and in the ilium and twisted until the graft was held firmly in place. Both iliac crests were used so that a parallel study could be made under the same conditions for each animal. The right iliac crest was injected with hyaluronidase and the left side remained untreated as a control. The number of injections and the amount of hyaluronidase per injection were varied in different rabbit groups. The animals were killed at 14 days, and the grafted areas were studied grossly and microscopically. Two rabbits died of an overdose of anesthetic and a serious wound infection developed in a third that rendered it useless for study. There remained therefore 26 grafted areas in 13 rabbits available for study.

As stated above, the animals were divided into various groups. Group 1 was a control series and consisted of three rabbits. No injections of any kind were given to this group. Group 2 contained four rabbits. Once a day for seven consecutive days, 150 TRU of hyaluronidase dissolved in 1 cc. of sterile saline was injected into the adjacent area on either side of the graft on the right iliac crest (making a total of 300 TRU of hyaluronidase per injection period). Group 3 consisted of four rabbits. A total of 300 TRU of hyaluronidase was injected in a similar manner around the right iliac graft every other day for a total of five injections. Group 4 contained one rabbit. A single injection of 300 TRU of hyaluronidase was made into the right ilium in a similar manner one hour after operation. No other injections were made. Group 5 contained one rabbit. An injection of 300 TRU of hyaluronidase was made into the right ilium in a similar manner 1 hour after operation and repeated once 48 hours postoperatively.

The animals in all five groups were killed at 14 days and the grafted areas studied grossly and microscopically. The histologic criteria for bone repair included (*a*) the presence of

‡ References 10, 11, and 12.

§ Wydase was supplied through the courtesy of Wyeth, Inc., Philadelphia. A TR unit is defined as the amount of hyaluronidase which will reduce the turbidity produced by 0.2 mg. of potassium hyaluronate in acidified horse serum to that produced by 0.1 mg. under assay conditions.

granulation tissue, (*b*) the presence of periosteal bone, (*c*) the presence of cartilaginous callus, (*d*) appositional new bone and replacement of old graft trabeculae with new bone, (*e*) the presence of trabeculae growing up from host marrow, and (*f*) marrow revascularization. Normal 14-day healing in the rabbit would show criteria (*a*) and (*c*) to be rated low and the remainder to be rated high.

In this series histologic study of the state of bone healing at 14 days based on the above criteria revealed no appreciable constant difference among the five groups. We would conclude that within the dosage range employed in this experiment there does not appear to be any constant deterrent action on bone repair attributable to hyaluronidase in the sections studied. From this it might be deduced that hyaluronidase is a safe agent to employ in any conditions in which the question of bone repair must be considered.

CLINICAL OBSERVATIONS

Soft-Tissue Trauma and Hemorrhage.—Trauma to soft tissues may result in considerable hemorrhage and swelling and if accompanied by fracture, as for example about the ankle joint, will often delay definitive treatment or complicate the result by increasing the likelihood of skin breakdown or wound infection.

Injection of 1,500 TRU of hyaluronidase into multiple sites about such a swollen ankle, with the use of an elastic bandage, has proved its efficacy on many occasions and will continue to do so if used early in adequate doses and if combined with pressure.

Soft-tissue hemorrhage alone may be a troublesome and sometimes disabling complication, particularly when surgical evacuation is impossible or impracticable. Hyaluronidase has proved itself to be a specific therapeutic agent where formerly none existed for this particular problem.

CASE 1.—G. H., a 42-year-old white man physician in good health, strained his left groin while playing tennis. He noticed the gradual onset of a throbbing ache in the left thigh and progressive inability to use the left leg without pain. Forty-eight hours after injury he found he could not arise from his bed because of severe pain in the left thigh and groin. When seen at home 60 hours after injury, there was a large tense diffuse hematoma in the left adductor mass. Hyaluronidase, 1,500 TRU dissolved in 3 cc. of 1% procaine and administered in doses of 1 cc., was injected into three sites in and about the diffuse hematoma and an elastic bandage applied. The throbbing pain disappeared during the next six hours. At 48 hours after injection the injured thigh was equal in size to the opposite side and the patient could move the extremity actively with very little pain. It was still tender to palpation at the site of injury. The patient resumed his regular schedule, including surgery, five days after the injection of hyaluronidase. The left thigh was quite ecchymotic but painless.

CASE 2.—S. A., a 23-year-old white Italian man with proved hemophilia, was admitted to the New York Orthopaedic Hospital with a large painful hematoma of 30 hours' duration beneath the palmar fascia of the right hand. When seen the palm was swollen and tense, and the fourth and fifth fingers were held in acute flexion, as in a typical Dupuytren contracture. Hyaluronidase, 500 TRU, dissolved in 1 cc. of 1% procaine was injected beneath the palmar fascia and an elastic bandage applied. Forty-eight hours after injection the palm was flat and the fingers could be actively flexed and extended through a complete range of motion.

Certain soft-tissue injury and hemorrhage may be followed by serious delayed effects. Myositis ossificans is a potential sequel to hemorrhage in any deep muscle, especially if the extravasation lies close to bone and in a closed fascial space. The familiar "Charley horse" of the athlete is always attended by the danger of a possible

myositis ossificans. The disorder is especially prone to develop after direct trauma to the quadriceps and brachialis muscles. The beneficial effects of hyaluronidase in several of these cases have been described in an earlier report.[1]

During the winter of 1952 to 1953 the opportunity of treating three additional patients with deep muscle hemorrhage presented itself. All three patients were medical students who sustained contused quadriceps with hemorrhage and hematoma formation while playing in a student basketball league. In all cases 1,500 TRU of hyaluronidase, dissolved in 6 cc. of 1% procaine and administered in doses of 2 cc., was injected in and about the hemorrhagic area and a pressure dressing applied. Tension and pain promptly subsided and the hematomas were rapidly dispersed. Discomfort did not recur, even after the anesthesia produced by the procaine had worn off. Although a startling amount of ecchymosis persisted, a rapid return to normal function occurred in all cases. Follow-up roentgen examination has failed to disclose evidence of myositis ossificans in any of the patients treated to date.

We have not proved experimentally that hyaluronidase will prevent myositis ossificans, nor is it possible to predict in which patients with soft-tissue trauma this complication will be most likely to develop. However, since hyaluronidase does cause early dissipation of hematoma, we believe that by injection of this enzyme-procaine mixture into every patient who seeks medical attention after muscle injury traumatic myositis ossificans may be prevented.

Impending Volkmann's Contracture.—An impending Volkmann ischemic contracture is a true orthopedic emergency and demands immediate treatment if disastrous consequences are to be averted. Dissipation of soft-tissue edema and hematoma is of utmost importance in prevention and treatment of the early Volkmann lesion. This complication most frequently follows supracondylar fracture of the humerus. The problem of a severe supracondylar fracture of the elbow and the ever-present concern for adequate reduction without circulatory impairment are common. The mixture of hyaluronidase and procaine offers an effective nonoperative method to relieve the subfascial tension caused by edema and hemorrhage with great rapidity. During the initial study period of this enzyme at the New York Orthopaedic Hospital, we had the opportunity of successfully treating with hyaluronidase two fresh supracondylar fractures that were complicated by impairment of the distal circulation due to subfascial tension caused by edema and hemorrhage.

Although less common than supracondylar fracture, other types of forearm trauma may be complicated by a Volkmann ischemic contracture.

Case 3.—A 5-year-old Negro boy was admitted to Jefferson Hospital 48 hours after his left forearm was caught in a wringer. When seen, the dorsum of the hand, the forearm, and the elbow were markedly swollen and tensely hard (Fig. 1). The fingers were held in a flexed position and any attempt at passive finger extension caused him great pain. The radial pulse was not discernible. Roentgenograms were negative for fracture. Hyaluronidase, 1,500 TRU dissolved in 3 cc. of 1% procaine and administered in doses of 1 cc., was instilled into the antecubital space, into the dorsal interosseous space, and into the volar interosseous space. A snug elastic bandage was applied from the finger tips to the axilla, and the extremity was elevated with adhesive traction. Twenty-four hours later the elbow and forearm were soft and the radial pulse was full and strong (Fig. 2).

Case 4.—A 14-year-old white boy, a proved hemophiliac, was admitted to Jefferson Hospital 36 hours after he slipped in the kitchen of his home and struck his right forearm. When seen, the elbow and forearm were stony hard and ecchymotic. The fingers were flexed and any motion

of them was extremely painful. There were scattered areas of hypesthesia over the dorsal and volar surfaces of the first three fingers. There was no palpable radial pulse. Roentgenograms were negative for fracture. Hyaluronidase, 1,500 TRU dissolved in 4 cc. of 1% procaine and administered in doses of 1 cc., was instilled into the antecubital space, into the dorsal interosseous

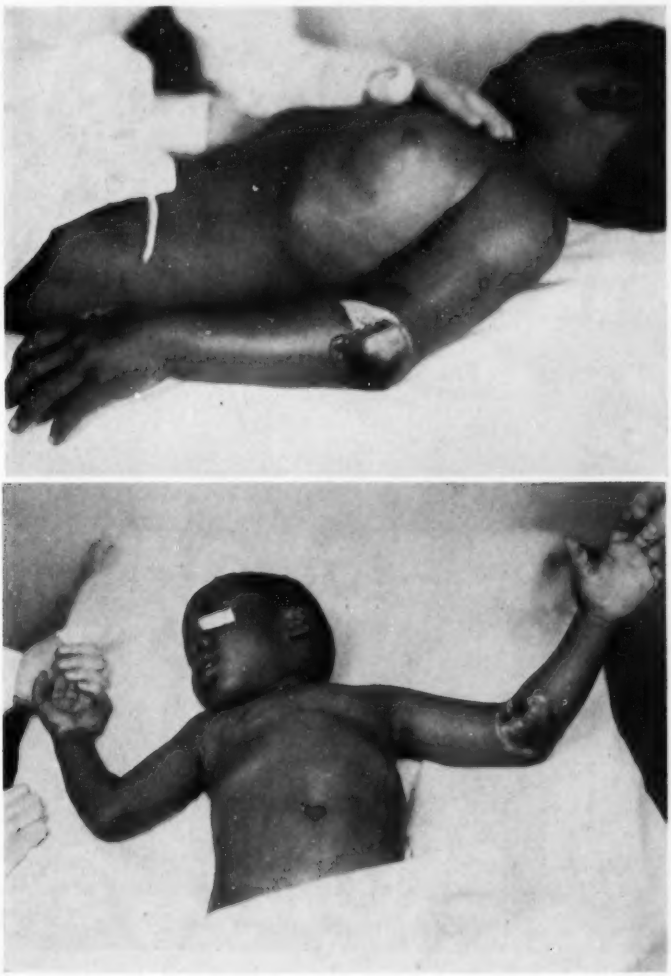

Fig. 1 (Case 3).—Photograph taken 48 hours after patient caught left arm in wringer. There was tense hard swelling of antecubital space and forearm; radial pulse not discernible.

space, into the volar interosseous space, and into the volar aspect of the wrist. A snug elastic bandage was applied from the finger tips to the axilla, and the extremity was elevated with adhesive traction. Twenty-four hours later the forearm was much softer and the radial pulse was palpable. At 48 hours the forearm was easily compressible and the radial pulse was equal to that of the other side. Good active finger motion was present, and sensation was about 75% intact.

Hemophilic Hemarthroses.—As we have reported elsewhere in greater detail,[15] hyaluronidase has found one of its most important uses in the treatment of acute hemophilic hemarthrosis. We believe the intra-articular injection of hyaluronidase combined with pressure to be the treatment of choice in these problem cases. Our

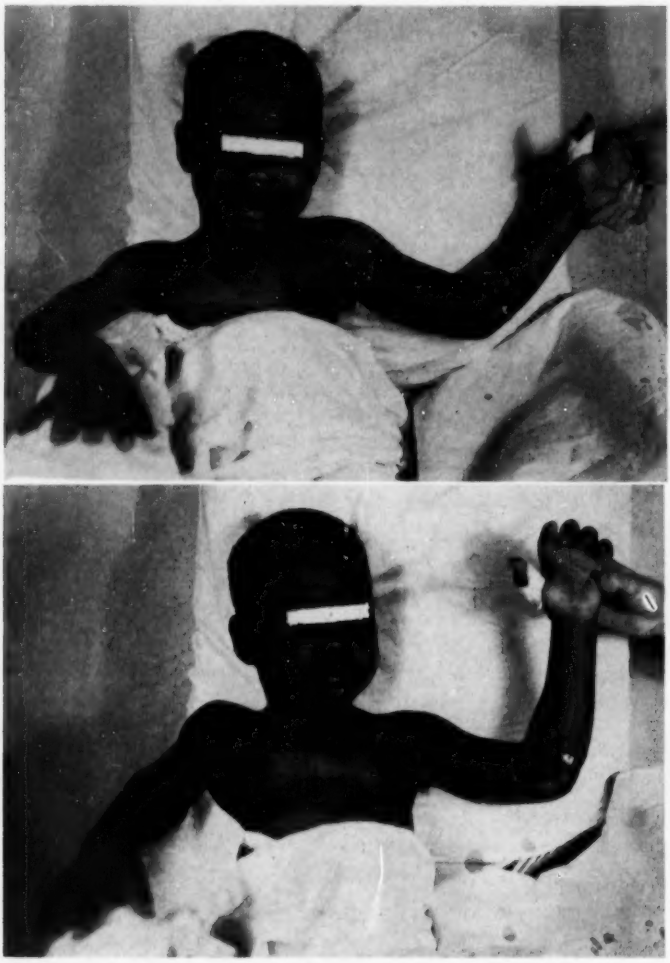

Fig. 2 (Case 3).—Photograph of patient taken 24 hours after hyaluronidase treatment. Antecubital space and forearm were soft; radial pulse, full and strong.

criteria for a successful result include complete absence of pain and restoration of the previous range of motion in 48 hours.

Early return of the customary range of motion precludes stiffening of the involved joint. Even more important, perhaps, is rapid restoration of a normal synovial fluid; thus reestablishment of adequate nutrition to the articular cartilage forestalls the degenerative changes that lead inevitably to crippling arthritis. Comparison of

the response to treatment by injection of hyaluronidase with the results obtained with earlier methods in the same joint in the same patient shows that recovery is not only more rapid after the injection of hyaluronidase but also more nearly complete.

The method is, in brief, as follows: When a hemophiliac is admitted to the hospital with acute hemarthrosis, the clotting time and other routine laboratory data are obtained. Plasma or fresh blood is given to lower the clotting time. The

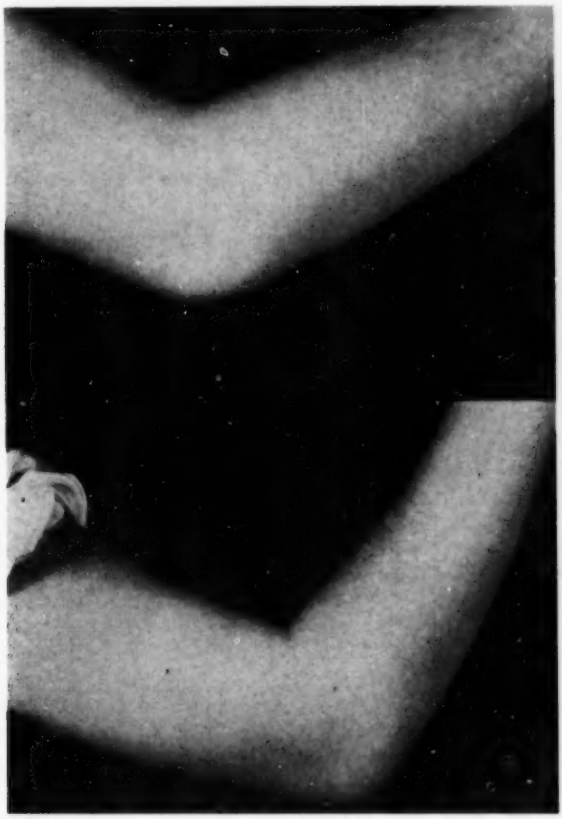

Fig. 3.—Acute hemophilic hemarthrosis of elbow of 24 hours' duration. Any motion painful. Maximum flexion and extension as shown.

joint is entered under aseptic precautions with a 20 gauge needle in the usual manner, and 3 to 4 cc. of the dark viscid blood is removed. Hyaluronidase, 1,000 TRU, is then mixed with 1% procaine in an amount equal to that of the blood withdrawn. This is injected into the joint, and a snug elastic bandage is applied. No treatment other than bed rest is necessary. When the patient is reexamined in 24 hours, pain is usually absent, and the range of motion is much greater. Occasionally a second injection is necessary. After 48 hours the patient can walk without pain or has free use of an upper extremity and may be discharged (Figs. 3 and 4).

It must be borne in mind that hyaluronidase is solely a dispersing agent. It has no coagulating effect and will not stop active bleeding in these joints. We rely on fresh whole blood or lyophilized plasma and clinical observation of the patient to determine when active bleeding has ceased. When this state has been reached, then hyaluronidase is used to clean out the joint cavity. It has been our experience that the best results are obtained if treatment is instituted during the 48 hours following subsidence of active bleeding. The results are equivocal in cases of hemarthrosis of

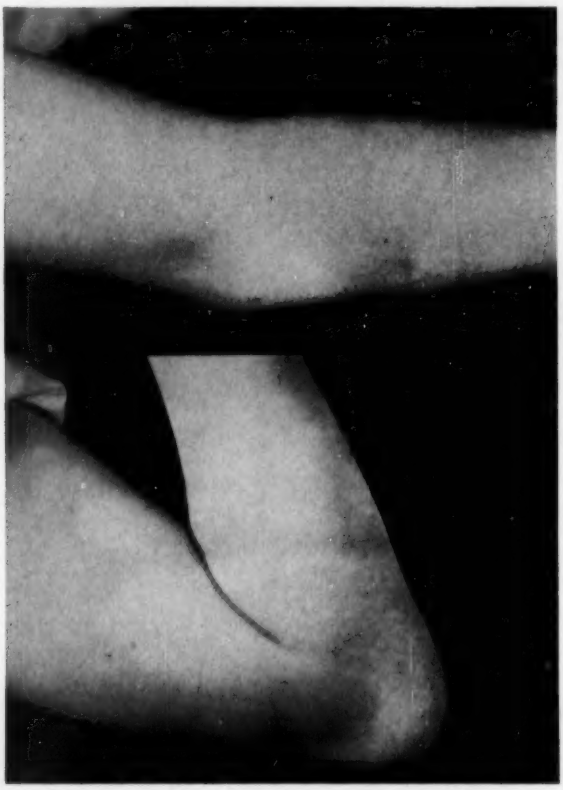

Fig. 4.—Forty-eight hours after hyaluronidase treatment. Painless motion. Maximum flexion and extension as shown.

one week or longer in duration. In none of our cases to date has the use of hyaluronidase shown any tendency to increase bleeding within the joint, nor have we had any trouble with bleeding from the needle puncture site.

At present the combined series from Presbyterian and Jefferson Hospitals comprise 29 acute hemophilic hemarthroses treated with hyaluronidase. In 10 cases the ankle joint was involved, in 10 the elbow, in 6 the knee, and in 3 the shoulder. In 26 cases pain was completely relieved and normal range of motion restored in 48 hours. In three patients the treatment failed for technical reasons in which the action of the enzyme had no part.

Seven patients suffering from large hemophilic hematomata with disabling symptoms also have been successfully treated with hyaluronidase in this series. We are convinced that the use of hyaluronidase is one of the most important advances in the management of acute hemophilic hemarthrosis and feel sure that early restoration of painless motion by this treatment will be a definite aid to prevention or postponement of permanent deformity.

Side-Actions.—No sensitivity reactions and no toxic manifestations attributable to hyaluronidase developed in any of the patients observed throughout the three-year evaluation period.

SUMMARY AND CONCLUSIONS

Evaluation of the usefulness of hyaluronidase in reducing soft-tissue swelling following injury over a three-year period has led to the following conclusions:

1. Hyaluronidase is a specific agent for definitive therapy of hematoma, hemarthrosis, and localized edema, as seen in orthopedic and traumatic surgery.

2. Experimental study on the influence of hyaluronidase in bone repair supports the conclusion that this enzyme alone in the usual clinical dosage does not deter bone healing.

3. Injection of hyaluronidase provides the most effective nonsurgical method for treatment of an impending Volkmann ischemic contracture. If the enzyme is used promptly in adequate dosage, operation will be unnecessary.

4. It is suggested that hyaluronidase if used early and in adequate dosage can prevent the development of myositis ossificans as a complication of soft-tissue trauma.

5. Hyaluronidase is the only effective agent known at present for definitive treatment of the hemarthroses and hematomata of hemophilia. It is the treatment of choice for acute hemophilic hemarthrosis.

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ABDOMINAL SURGERY IN THE ELDERLY PATIENT

Review of 106 Selected Cases of Biliary-Tract, Stomach, and Bowel Lesions

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THE VERITABLE plethora of recent literature on the subject of geriatric surgery is ample evidence that increasing attention is being focused upon the aged of our population. This clear-cut trend has its origin in the fact that the number of persons in the upper age brackets has been increasing since the turn of the century. Groom [1] has pointed out several interesting facets of population changes in the United States. Between 1930 and 1940 the entire population increased by only 7%, whereas there was an increase of 35% in the number of persons 65 to 74 years of age and an increase of 38% in those more than 75 years of age. It is estimated that 40% of the population will be more than 45 years of age by 1980, while the group over 65 years of age will represent 14% of the total population, or 22,000,000 persons. Inasmuch as increasing numbers of the elderly will require surgical treatment, it is important to analyze, evaluate, and report the results in this particular group of patients.

TABLE 1.—*Material Studied*

Type of Cases	Patients, No.	Operations, No.
Biliary Tract	25	28
Colon	49	51
Gastric	32	32
Total	106	111

MATERIAL STUDIED

This presentation is a review of 106 consecutive patients 70 years or older who were subjected to major abdominal surgery on the private and ward services of one of us (R. C.) at the Mount Sinai Hospital for an 11-year period from Jan. 1, 1941, to Dec. 31, 1951 (Table 1). This series does not represent all who were treated. It is a selected group in which the magnitude of the operation performed on the biliary tract, stomach, and colon represented a maximum challenge to body reserves. It was felt in this way that surgical tolerance could be most profitably analyzed. Several patients were operated upon more than once. Preliminary cecostomies and colostomies are not included in the figures. While it is true that the inclusion of such cases would have drastically altered the mortality figures, it is obvious it would have served neither the purpose nor value of this study.

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PREOPERATIVE EVALUATION AND PREPARATION

Old age per se is rarely a contraindication to operation. The fact that survival has been maintained in spite of all previous vicissitudes speaks for a physical constitution which must possess vital organs of good functioning capacity. It is scarcely necessary to remind physicians and nurses that the care of the aged requires sympathy, tact, and understanding. They welcome reassurance, and often an octogenarian will brighten when told he will tolerate operation. The elderly are annoyed and frightened by trifling details. They do not enjoy restrictions of their established habits, and the latter should be interefered with as little as is compatible with proper professional care.

Many elderly patients are in a relatively poor state of nutrition. While less food is required because of reduced activities and lower levels of metabolism, many either because of ignorance or economy suffer from the effects of improperly balanced or inadequate diets. Then, too, prolonged infection, cancer, and bleeding may further deplete stored glycogen, fat, available protein, and vitamins. All these possess important nutritional values, especially in times of stress and starvation. Adequate oral alimentation, high in protein and rich in vitamin C essential for wound healing and resistance to infection, and vitamin B complex, a participant in many cellular reactions, are generally accepted as excellent preoperative preparation. Often this is impracticable because of the necessity of urgent surgery, and therefore parenteral therapy, including blood transfusions, is frequently required to correct rapidly severe anemia, hypoproteinemia, and vitamin deficiencies and electrolytic imbalances.

There is a definite ceiling on the rate of both hemoglobin synthesis and red blood cell regeneration.* A defect in hemoglobin synthesis has been identified in chronic infections [4] and in malignant disease,† and it is mandatory that hemoglobin deficiency be corrected preoperatively.

The existence of certain priorities for the selective replacement of specific body proteins has been established: (1) hemoglobin,[7] (2) tissue protein and plasma globulin, and (3) plasma albumin.[8] If hemoglobin deficits are corrected prior to surgery, then such protein reserves as are available can be utilized for wound healing.

The diminished reserve and magnitude of associated disease in these patients leave but little margin for error. Many suffer from hypertension, congestive heart failure, and diabetes. Their preoperative preparation calls for close cooperation between the surgeon and the internist. Electrocardiograms are always done in addition to a routine complete blood examination, including an estimation of the blood proteins, urea, chloride, sodium, and potassium. It is highly essential that any clinical imbalances and fluid disturbances are corrected prior to operation.

A word of caution is in order regarding routine hemoglobin and blood protein determinations. It has been adequately demonstrated [9] that no true correlation exists between hemoglobin as measured in grams per 100 ml. and the total circulating hemoglobin mass or between the hematocrit reading and the circulating hemoglobin deficit. Similarly, there is no correlation between plasma proteins as

* References 2 and 3.

† References 5 and 6.

measured in grams per 100 ml. and the total circulating plasma proteins or plasma protein deficit. The concentration values commonly available on the hospital chart are significant only when the blood volume, including all its components, is normal. Calculations of total blood volume, plasma volume, circulating hemoglobin, and circulating protein deficits are the only satisfactory bases for acute replacement. Bosch and his associates [9] demonstrated that reliance on routine hemoglobin and serum protein determinations resulted in inadequate blood replacement, as compared with the requirements dictated by blood volume determinations.

The problem at hand may well be compared to the "chronic shock" syndrome described by Lyons and his co-workers on the basis of observations upon injured soldiers in World War II.‡ The chronically ill patients studied by this group presented a picture characterized by (1) loss of weight, (2) diminished tolerance for loss of blood during necessary surgical procedures, (3) delayed or faulty healing of wounds, (4) retarded convalescence, (5) reduced blood volume with a deficiency in total circulating red blood cell mass marked by constriction of the plasma volume, and (6) increased interstitial fluid volume.

Unfortunately the determination of blood volume is neither simple nor inexpensive; consequently, such studies upon all elderly patients will not be feasible in the average hospital. Practically, however, whole blood transfusions in sufficient quantities should be administered to restore the total circulating red blood cell mass. But in this repletion of blood volume the cardiovascular status of the patient must be carefully observed so that right heart failure and pulmonary edema do not occur.

While general principles of preoperative preparation have been discussed, there are other preparative measures necessary under specific conditions.

Patients with stenosis at the pyloroduodenal junction secondary to ulcer disease or carcinoma have large dilated atonic edematous stomachs filled with retained secretions and ingested food. Unless the stomach is completely evacuated these patients are in danger of aspiration during anesthesia. In our Clinic, several days are devoted to morning and evening gastric lavages with a large Ewald tube utilizing several quarts of tap water at a temperature of 105 F. The volume of retained gastric contents is carefully noted at each aspiration, and operation is not usually undertaken before the retention has fallen to 100 cc. or less. This practice has diminished the danger of aspiration and of contamination of the peritoneal cavity during surgery, and edema of the stomach wall has been lessened, resulting in better healing.

The utmost attention must be paid to the frequently severe hypochloremia, hypopotassemia, alkalosis, and dehydration which result from vomiting due to long-standing pyloric obstruction aggravated by the leeching effect of gastric lavages. As much as 2,000 to 3,000 cc. of isotonic sodium chloride solution may be required daily for correction of the electrolyte deficits. Since vomiting results in a greater loss of chloride than of sodium, care should be taken not to administer excess sodium. A 2% solution of ammonium chloride will be effective in this regard, since it will furnish chloride without additional sodium. Potassium chloride is added to

‡ References 10 and 11.

the solutions as required. Occasionally, markedly depleted patients require hypertonic sodium chloride. However, caution must be exercised to avert overcorrection when using this type of fluid.

Daily determinations of serum sodium, chloride, potassium, carbon dioxide-combining power, and urinary output will provide the necessary information for variations in the amounts and types of fluid and electrolytes required during the preoperative period.

The oral use of sulfonamides and antibiotics in the preoperative period prior to bowel resection is now generally accepted. It need only be pointed out that the use of sulfonamides demands an increase in the intake of certain vitamins. Four to six days of succinylsulfathiazole (Sulfasuxidine) administration results in marked diminution of the thiamin output in the feces unless the thiamin intake in the diet is greatly augmented.[12]

The physiologic requirement of vitamin K is met in large part by the absorption of vitamins synthesized in the bowel. Reduction of the bacterial population of the colon results in a definite reduction in the synthesis of vitamin K, which may result

TABLE 2.—*Anesthesia (All Cases)*

Anesthetic Agent	Operations, No.
Spinal	47
Cyclopropane	27
Nitrous oxide, oxygen, ether	15
Ethylene	12
Avertin	4
Local	6
Total	111

in hypoprothrombinemia and hemorrhage at the suture line.[13] This effect can be overcome by the oral administration of vitamin K during the preoperative period.

Daily purgation and colonic flushes are utilized to clear the colon of gross fecal content. If a preliminary cecostomy or colostomy has been performed, then these stomata are irrigated as well.

ANESTHESIA

Preanesthesia medication was always individualized. Doses were generally two-thirds to one-half of those used in younger adults, and, because of the slower absorption in the aged, the premedication was administered earlier. Its effect usually lasts longer owing to delayed excretion and detoxification. In the presence of mental confusion due to advanced cerebral arteriosclerosis or other causes the use of barbiturates and of scopolamine hydrobromide, which are likely to cause excitement and restlessness, was avoided.

We agree with Lundy [14] that the reduction of anesthesia risk can best be accomplished if the anesthetist is skilled, regardless of the agent employed. The various anesthetic modalities employed are summarized in Table 2.

Spinal anesthesia was utilized without any complications per se in 47, or 42.3%, of the cases. It has definite advantages. It affords unsurpassed relaxation, giving good exposure and easy access to all intra-abdominal viscera. It improves the cir-

culation of the lower extremities, and a beneficial diuretic effect has been described. When there is a recent history of congestive heart failure, spinal anesthesia is considered best tolerated, since it produces peripheral vasodilatation which acts as a bloodless phlebotomy.[15] We are aware that high spinal anesthesia paralyzes the vasoconstriction of the splanchnic bed and may produce marked fall of blood pressure that may be difficult to correct, especially in the presence of hypertension and advanced arteriosclerosis.[16] In our experience, however, the competent anesthetist has avoided these pitfalls.

General anesthesia was utilized in 58, or 52.3%, of the cases. In recent years, curare has been the supplemental muscle relaxant used concomitantly in most of the cases having general anesthesia.

In six patients who were particularly poor risks, local and regional anesthesia were employed, since these agents apparently cause the minimal interference with

TABLE 3.—*Pathology in Biliary-Tract Cases*

		Cases, No.
Chronic cholecystitis in gall-bladder remnant		1
Chronic cholecystitis, cholelithiasis		7
Chronic cholecystitis, cholelithiasis, choledocholithiasis		8
Choledocholithiasis		2
Acute cholecystitis, cholelithiasis		6
Carcinoma of gall bladder		2
	Total	26 *
Associated diseases		4
Biliary cirrhosis	1	
Acute cholangitis	1	
Chronic pancreatitis	1	
Cholecystoduodenal fistula	1	

* One patient had two operations.

the function of the organism as a whole. Aged patients, because of their decreased reflex irritability and poor sensitivity, seem to tolerate these forms of anesthesia well.

SURGICAL INDICATIONS, SURGICAL PATHOLOGY, AND PROCEDURE

If the operative mortality and postoperative morbidity are to be kept at a minimum, all general surgical principles must be carefully observed, and the operative procedures should be expeditiously performed with minimal trauma.

The specific surgical indications, operative findings, and procedures employed will now be briefly summarized in the various groups.

Biliary-Tract Surgery (Tables 3 and 4).—Twenty-five patients comprised this group. There was a total of 28 operations, three patients undergoing two operations. Their ages ranged from 70 to 85 years (average 73 years). Seven were men and 18, women. These figures are in general agreement with past observation of the preponderance of gall-bladder disease in women.

Several patients because of repeated severe attacks of gallstone colic demanded relief. Biliary-tract infections manifested by the Charcot syndrome which did not respond to adequate doses of antibiotics were treated surgically, as were cases of

acute cholecystitis with or without a mass. Gangrene of the gall bladder with early perforation and a generalized peritonitis is a frequent occurrence, especially in the aged diabetic patient.

A wide spectrum of biliary-tract disorders was encountered. The gall bladders which invariably contained calculi were usually shrunken vestiges embedded deeply in the liver and surrounded by multiple adhesions, resulting in a distortion of the contiguous anatomy. Cholecystectomy which was the treatment of choice was performed in 21 cases. The recognized indications for choledochostomy were followed, and the common duct was explored in 13 cases, with or without an associated procedure on the gall bladder. In one case an impacted ampullary calculous was removed by transduodenal choledochostomy. The only case in which choledochostomy failed to reveal calculi was that of a 72-year-old woman who had had a cholecystectomy for calculi 32 years previously. Because of persistent right upper quadrant pain, reexploration was done. A chronically inflamed gall-bladder stump without stones was found. The pancreas was normal.

TABLE 4.—*Analysis of Surgical Procedures in Biliary-Tract Cases*

	Operations, No.
Cholecystectomy (primary)	10
Cholecystectomy (secondary)	2
Cholecystectomy (primary) with common duct exploration	5
Cholecystectomy (primary) with common duct exploration and closure of cholecystoduodenal fistula	1
Cholecystectomy (secondary) with common duct exploration	2
Cholecystectomy (primary) with common duct exploration and duodenotomy	1
Common duct exploration	2
Cholecystostomy	3
Cholecystostomy with common duct exploration	2
Total	28

There were six cases of acute cholecystitis. In four, cholecystectomy was performed uneventfully. One of these patients, 79 years of age, required reoperation two years later because of recurrent right upper quadrant pain. The common duct, which contained gravel and sludge, was drained, and recovery ensued. In the remaining two patients, cholecystostomy was instituted.

There were two cases of carcinoma of the gall bladder associated with cholelithiasis. One of these patients who was desperately ill underwent exploration during an attack of acute cholecystitis, and, because of the density of adhesions, the thick-walled gall bladder which contained a solitary cholesterol stone was simply drained. Exploration was again performed on the 27th postoperative day because of persistent spiking temperature. At this time carcinomatous metastases in the right lobe of the liver and extension to the porta hepatis and gastrohepatic ligament were found. Death occurred two days later.

The second case of carcinoma of the gall bladder was seen during a subacute stage with jaundice. At operation the gall bladder was thick walled, distended with mucopurulent fluid, and contained many facetted stones. Cholecystostomy and choledochostomy were performed. Convalescence was uneventful. She underwent exploration six months later for recurring right upper quadrant symptoms, at

which time the gall bladder was thick walled and its posterior wall stony hard. The common duct was normal. The excised gall bladder was the seat of an infiltrating adenocarcinoma.

In view of the fact that the pathology was not suspected in these two cases, it might be advisable to remove a portion of the gall bladder for pathologic examination in all cases of acute cholecystitis in which cholecystostomy is performed. While

TABLE 5.—*Pathology in Colon Surgery Group*

Lesion	Cases, No.
Carcinoma	
Cecum	12
Hepatic flexure	2
Transverse colon	2
Descending colon	4
Sigmoid	10
Rectosigmoid	4
Rectum	13
Total	47
Lymph nodes involved	9 (19.2%)
Lymph nodes not involved	38 (80.8%)
Villous adenoma of splenic flexure	1
Acute diverticulitis of splenic flexure	1
Coexisting adenomatous polyps	15 (31.9%)
Coexisting submucous lipoma	2

TABLE 6.—*Analysis of Surgical Procedures in Colon Cases*

		Operations, No.
Ileocolic resection with ileotransverse colostomy		13
Transverse colon resection with end-to-end anastomosis		1
Splenic flexure resection with end-to-end anastomosis		2
Descending colon resection with end-to-end anastomosis		3
Sigmoid resection with end-to-end anastomosis		4
Anterior resection with end-to-end anastomosis		6
Combined abdominoperineal resection (Miles)		10
Two-stage abdominoperineal resection (in one patient)		2
Lockhart-Mummery: sigmoid colostomy and perineal resection		1
Obstructive resection		8
Transverse colon	1	
Descending colon	1	
Sigmoid colon	6	
Exploratory laparotomy		1
Total		51

the present series is too small to carry statistical validity (2 cases of carcinoma in 23 cases of cholelithiasis), the relation of chronic gallstone irritation to carcinoma may be significant.

Colonic Surgery (Tables 5 and 6).—In this group 49 patients underwent 51 operations upon the colon. Their ages ranged from 70 to 83 years, with an average of 73 years. There were 28 men and 21 women.

The surgical procedures performed depended upon the degree of obstruction, as evidenced by x-ray examination, and on the locations of the lesions. Patients

with acute intestinal obstruction were decompressed after a short period of hydration by colostomy or cecostomy prior to definitive resection. No time was wasted by the introduction of a long intestinal tube.

Right colonic neoplasms were treated by ileocolic resection and ileotransverse colostomy in 13 cases. Segmental colonic resection was performed in 24 cases in which the tumor involved the left colon. In recent years in most cases a primary resection with end-to-end anastomosis with complementary colostomy or cecostomy has been preferred to obstructive resection even in the poor-risk obese patient with chronic obstruction. We have continued to perform palliative resections in these cases with obstruction in spite of hepatic metastases.

A few patients with carcinoma of the rectum who were especially poor risks were treated by fulguration. Abdominoperineal resection was done in 11 patients. We have tended to veer away from anterior resection for carcinoma of the rectum because of the high rate of recurrence.[17] However, we feel it may be indicated in some elderly patients, especially those affected by the infirmities of time, who often live alone and have defective vision and senile psychoses. This group is utterly unable and frequently unwilling to cope with the demands of even a well-functioning colostomy. It would appear advisable to sacrifice some of the principles of cancer

TABLE 7.—*Pathology in Gastric Surgery Group*

	Cases, No.
Carcinoma of stomach	15
Lymphosarcoma of stomach	1
Benign gastric ulcer	8
Duodenal ulcer	8
Total	32

surgery to allow the more infirm of these patients to spend their remaining months or years with a normal-functioning anus. Anterior resection was therefore done in six instances.

All the resected lesions were carcinoma except one which was an obstructing diverticulitis. The tumors were located in various parts of the colon, but 57% occurred in the sigmoid, rectosigmoid, and rectum. Coexisting adenomatous polyps were present in 31.9% of these cases, a fact which emphasizes the importance of polyps as precursors of carcinoma.[18]

The incidence of lymph node metastasis presents an interesting side-light in this group of patients. Of the 47 carcinomas, 38, or 80.8%, failed to show lymph node involvement. Only 9, or 19.2%, of the nodes were involved by metastatic carcinoma. Gilchrist and David [19] in a review of 200 cases of carcinoma of the colon and rectum noted 62.5% lymph node metastasis, and Grinnell,[20] 47% in a series of 322 specimens. Accurate comparison with the present small series of elderly patients of course cannot be made, but it is of interest to note the low rate of lymph node metastasis as compared with series covering all age groups. We can only speculate on the relative low incidence of lymphatic spread in these patients past 70 years of age.

Gastric Surgery (Tables 7 and 8).—There were 32 patients in this group. Their ages ranged from 73 to 84 years (average 73 years). The cases were equally divided between men and women. Most cases of severe hemorrhage, either of gastric

or of duodenal origin, were treated conservatively. We feel now that this was a mistake. While the bleeding ceased spontaneously in many patients, others died of hemorrhage or of the eventual results of blood loss, such as myocardial infarction. We are now convinced that emergency surgery should be performed, especially if hemoglobin values are not maintained by adequate transfusions of blood during the first 48 hours. Eight cases of duodenal ulcer with intractable pyloric obstruction due to scarring were operated upon. A gastroenterostomy was performed with a complementary infradiaphragmatic vagotomy in those cases in which gastric analysis revealed elevated values of free hydrochloric acid. In one case of a gastric ulcer which was juxtaesophageal in position and proved to be benign, a Madlener procedure was performed. We appreciate the criticism which may be leveled at the possibility of mistaking a benign ulcer for carcinoma, but this is no worse than performing a total gastrectomy for a benign ulcer in patients over 70 years of age. It might be well to mention the advantages of complementary jejunostomy for alimentation in long-standing cases of pyloric obstruction with marked gastric dilatation, attendant debility, anemia, and hypoproteinemia.

TABLE 8.—*Analysis of Surgical Procedures in Gastric Cases*

Type of Operation	Operations, No.
Subtotal gastrectomy	17
Subtotal gastrectomy and feeding jejunostomy	1
Total gastrectomy	2
Anterior gastroenterostomy	1
Posterior gastroenterostomy	4
Posterior gastroenterostomy and bilateral infradiaphragmatic vagotomy	2
Posterior gastroenterostomy and feeding jejunostomy	2
Kelling-Madlener operation	1
Anterior gastroenterostomy with exclusion of carcinoma of distal stomach	2
Total	32

There were 15 cases of gastric carcinoma. In the majority of these the lesions were large, either by palpation or x-ray examination, and evidently had been present for a long time before causing symptoms. The size of the lesion does not bespeak for inoperability, and often a carcinoma which is locally inoperable may be treated palliatively. In all cases exploration was done unless ascites, demonstrable liver metastases, or a palpable rectal shelf pointed to inoperability.

A high subtotal gastrectomy with removal of the omentum and glands was performed in 11 cases. Total gastrectomy was done in two patients because of the extensive involvement of the stomach without evidence of distant metastases. Two locally unresectable pyloric and antral lesions fortunately could be excluded with continuity restored by gastroenterostomy, a procedure which affords a fair degree of palliation.

POSTOPERATIVE CARE AND COMPLICATIONS

Careful attention to every detail yields worth-while dividends. A complication in the postoperative period of an elderly patient is surmounted only with difficulty, and, if not fatal, results in a much greater prolongation of hospitalization than obtains in younger persons.

The postoperative complications listed in Table 9 indicate that abdominal and respiratory infections constitute the major hazards in this type of surgery. These have been reduced in number and consequence in the latter years of this study following a better understanding of fluid balance, of early ambulation, and of antibiotics.

TABLE 9.—*Postoperative Complications (All Cases)*

		Cases, No.
Cardiovascular system		15
Congestive heart failure	6	
Coronary insufficiency	1	
Auricular fibrillation	3	
Auricular flutter	2	
Auricular and ventricular premature contractions	1	
Bradycardia	1	
Thrombophlebitis	1	
Respiratory system		20
Bronchopneumonia	10	
Atelectasis	3	
Pulmonary edema	4	
Pulmonary infarct	2	
Pleurisy	1	
Nervous system		1
Cerebral accident	1	
Abdomen		23
Wound infection	6	
Wound dehiscence	3	
Fecal fistula	2	
Biliary fistula	3	
Duodenal stump fistula	1	
Peritonitis	1	
Paralytic ileus	1	
Intestinal obstruction	1	
Acute cholecystitis	1	
Liver abscess and thrombophlebitis of portal vein	1	
Necrosis of colostomy	1	
Gastric retention	2	
Genitourinary tract		4
Oliguria	2	
Ureteral obstruction	1	
Acute epididymitis	1	
Miscellaneous		8
Anemia	3	
Jaundice	1	
Arteriosclerotic gangrene of all toes of left foot	1	
Psychosis	1	
	Total	71

Fluid and electrolyte balance must be carefully controlled. The temporary disturbances of electrolyte metabolism following surgery are now well known.§ The usual alterations include marked reduction in the output of sodium. Our patients receive salt-free fluids parenterally in the immediate postoperative period, and saline is administered later where indicated and in limited quantities, allowances being made for losses in urine, perspiration, intestinal and gastric suction, vomiting, and draining fistulae.

§ References 21 and 22.

Three cases in this series were given excessive fluids, resulting in pulmonary edema and cardiac failure. These patients were treated between 1941 and 1944, when the dangers of excess salt and fluid were not fully appreciated.

In addition, the dangers of excessive salt-free dextrose administered at the time of surgery and in the immediate postoperative period should be appreciated. Zimmerman and Wangensteen [23] reported 17 cases of postoperative convulsive seizures in elderly patients characterized by dilution of the extracellular electrolytes. In most instances, strongly positive fluid balance was demonstrable by the intake and output data and the change in body weight. They emphasize that these patients are extremely sensitive to water excess for 48 hours after surgery. They recommend that no more water be administered than is necessary to cover predictable fluid losses and assure adequate urinary output. In our experience, 1,500 to 2,000 cc. of 5% dextrose in distilled water has been adequate for most elderly patients during the first 24 hours postoperatively.

TABLE 10.—*Comparative Mortality, Early Recent Cases*

Group	Deaths, No.	Operations, No.	Operative Mortality, %	Patients, No.	Patient Mortality, %	
Biliary tract	0	10	0	8	0	1941 to 1946
Colon	10	26	38.5	24	41.7	
Gastric	2	17	11.8	17	11.8	
Total	12	53	22.6	49	24.5	
Biliary tract	1	18	5.6	17	5.9	1947 to 1951
Colon	2	25	8.0	25	8.0	
Gastric	0	15	0	15	0	
Total	3	58	5.2	57	5.3	

The average period of bed rest was three days. The great majority of patients were out of bed on the first postoperative day, and many were mobilized even the day of operation. If possible, they are urged to walk at the very onset, for in this way pulmonary ventilation is enhanced, venous stasis averted, and urination encouraged. A weak patient slumped in a chair is better off supine in bed.

MORTALITY

Operations upon the biliary tract and stomach are rather well tolerated. The formidable mortality of colonic surgery noted in the first six-year period has been reduced in the second five-year period (Tables 10 and 11).

There was 1 death in the biliary-tract group, 2 in the gastric, and 12 in the colon group, a total of 15 deaths, a patient mortality of 14.2% for the entire group. When the series is divided into the early period (1941 to 1946) and into the later period (1947 to 1951), the operative patient mortality of 24.5% in the first period falls to 5.3% in the second period.

Three deaths were caused by cardiac failure, pulmonary edema, and bronchopneumonia. Pulmonary infarct, peritonitis, thrombophlebitis of the portal vein, cerebral hemorrhage, and pylonephritis each contributed one death, while one patient died of undetermined causes.

Study of the deaths occurring in the first five-year period (1941 to 1946) reveals that 7 out of the total of 12 might possibly have been saved had the various components of our present-day armamentarium been available. Cases 4, 5, and 6 were unequivocally the result of excessive salt and water intake in the light of our recently acquired knowledge. Case 12 following an obstructive resection of the colon died of thrombophlebitis of the portal vein and liver abscess proved at autopsy. It is conceivable that modern antibiotics might have turned the tide in favor of recovery.

TABLE 11.—*Summary of Deaths After Operation*

Case	Sex	Age, Yr.	Findings	Operation	Cause of Death
1	F	73	(1) Acute cholecystitis, solitary gallstone; (2) one month later, carcinoma of gall bladder with extension to porta hepatis and rt. lobe of liver	(1) Cholecystostomy; (2) cholecystectomy	Cardiac failure
2	M	70	Adenocarcinoma of upper descending colon	Resection of splenic flexure with end-to-end anastomosis	Cardiac failure; ? peritonitis
3	F	84	Adenocarcinoma of stomach	Subtotal gastrectomy, feeding jejunostomy	Cardiac failure
4	M	70	Adenocarcinoma of rectum	Abdominoperineal resection	Pulmonary edema
5	F	70	Adenocarcinoma of cecum	Ileocolic resection	Pulmonary edema
6	M	72	Adenocarcinoma of rectum	Abdominoperineal resection	Pulmonary edema
7	M	73	Adenocarcinoma of descending colon	Preliminary cecostomy followed by resection of descending colon with end-to-end anastomosis	Bronchopneumonia; ? peritonitis
8	M	71	Adenocarcinoma of rectosigmoid	Sigmoid spur colostomy followed by anterior resection	Bronchopneumonia *
9	M	79	Adenocarcinoma of transverse colon	Obstructive resection	Bronchopneumonia
10	M	72	Scirrhous carcinoma of stomach	Total gastrectomy, feeding jejunostomy	Pulmonary infarct
11	M	73	Adenocarcinoma of cecum	Ileocolic resection	Peritonitis
12	F	72	Adenocarcinoma of sigmoid descending colon	Preliminary cecostomy followed by obstructive resection	Abscess of liver *; thrombophlebitis of portal vein
13	M	72	Adenocarcinoma of rectum	Abdominoperineal resection	Cerebral accident
14	F	72	(1) Adenocarcinoma of rectum; (2) postop. intestinal obstruction due to adhesions	(1) Abdominoperineal resection; (2) lysis of adhesions, 6th postop. day	Pyelonephritis *; hydronephrosis
15	F	71	Adenocarcinoma of rectum	Two-stage abdominoperineal resection	Undetermined

* Autopsy obtained.

Severe bronchopneumonia resulted in the unfavorable outcome in Cases 8 and 9. The former failed to respond to intravenous administration of sulfathiazole, while sulfadiazine was ineffective in the latter. Penicillin had not yet appeared on the scene.

Case 11 developed postoperative peritonitis, which progressed in spite of the use of penicillin and sulfadiazine. Chlortetracycline (Aureomycin) and oxytetracycline (Terramycin) were years away.

Case 15 died of undetermined causes, and the fatal outcome in Cases 3, 10, and 13 was unrelated to advances in therapy.

Case 14 died of a kidney infection. The left ureter was inadvertently occluded by the suture closing the peritoneal floor during the performance of abdominoperineal resection for carcinoma of the rectum.

Of the three deaths occurring in the period 1947 to 1951, two cases (Cases 1 and 2) were due to cardiac failure, with peritonitis a contributing factor in Case 2. The third death, Case 7, was due to bronchopneumonia and a probable peritonitis which did not respond to chlortetracycline, penicillin, and streptomycin.

EVALUATION OF SURGICAL RISK

One of the aims of this study was to determine if possible the probability of survival of a given elderly patient with a known disease and associated complications such as hypertension, coronary artery disease, congestive heart failure, etc.

Renal Disease.—None of the 15 fatalities exhibited evidence of renal damage preoperatively.

Diabetes Mellitus.—Three patients in this group had diabetes, but since they were never out of control and were not severe cases of diabetes, the high mortality for diabetic patients (three out of nine) appears coincidental.

Previous Coronary Occlusion.—One of the fatal cases fell into this category, representing 33.3% of those so classified preoperatively. This patient is one of those listed as having died of pulmonary edema due to overloading with salt and water.

Congestive Cardiac Failure.—None of the fatal cases was in this category. Two patients in this series presented this preoperative complication, and both did well. One of these was in failure at the time of admission to the hospital and was treated with the usual regimen before surgery. A patient in congestive failure is generally considered a poor surgical risk. Actually, if this complicating medical state is properly managed, the risk of surgery is not unduly great.

Hypertension.—There were 33 patients classified as having hypertension preoperatively. There were three deaths evidently not directly related to the elevated blood pressure and associated cardiovascular disease, since two patients died of bronchopneumonia and the third of peritonitis. It does not appear therefore that hypertension per se constitutes a great risk.

Obesity.—Fourteen patients were classified as obese in the preoperative evaluation. Four (29.6%) died. It has long been the contention of one of us (R. C.) that obesity constitutes a major hazard.

Anesthesia.—Seven of the fatal cases had spinal anesthesia; four had cyclopropane, two ethylene, one tribromoethanol (Avertin), and one local. This distribution of anesthetics parallels the frequency of utilization in the entire series; consequently, no particular modality can be assigned a lethal potentiality.

Nature of Operation.—Surgical procedures of the colon carry a patient mortality of 8.0% (recent years), those on the biliary tree 5.9%, while surgery of the stomach is well tolerated, with no mortality in this group in recent years. The nature of the lesion and the type of surgical procedure required for its correction appear to us to be the most significant of factors in mortality.

SUMMARY AND CONCLUSIONS

Geriatric surgery is acquiring increasing importance as our elderly population enlarges.

The pathology and treatment and pre and postoperative care of 106 selected patients 70 years and older are presented.

Old age is not a contraindication to surgery. Sex, anesthesia, hypertension, previous congestive heart failure, and technical considerations are not important mortality factors. The presenting disease and the surgical procedure required to correct it are significant mortality factors, as is also obesity.

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PATHOLOGY OF THE APPENDIX

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THE DISEASED vermiform appendix is a common but deadly acute surgical entity. Meyer, Requarth, and Kozoll [1] found an over-all mortality which had been reduced from 7.6 to only 4.3% in 5,543 cases reviewed. Collins,[2] in his review of 12,813 consecutive appendectomies, reports a similar mortality. The mortality and morbidity of appendicitis persists in spite of better diagnostic acumen and therapeutic measures, including intragastric suction, intravenous fluids, and whole blood and plasma transfusions; and chemoantibiotic therapy, which have been vigorously applied in the treatment of this disease.

Justifiably, this "microcosm in which all the various diseases to which its component anatomical elements are liable in other parts of the body may occur," [3] is the subject of recent intensified study and research.[4] Appendiceal disease is not to be held lightly, nor in contempt. Collins stresses that only the uninitiated views an appendectomy as mere "routine"; "the experienced surgeon knows that an operation for acute appendicitis can be extremely simple or one that will tax to the utmost all of his previous skill and years of surgical judgment." [2]

This paper is offered as a contribution toward a better understanding of the problem as it presents itself clinically; in the operating room as a specific entity, or *en passant;* in the examination of the surgical specimens, or at autopsy.

For the purposes of this study 377 cases were reviewed. Histories were perused, operative procedures noted, and the clinical course observed whenever possible. The microscopic sections were instructive. One series * were invariably longitudinal sections; all other cases † were cross sectioned for microstudy. One case, a carcinoid tumor, was obtained from the Illinois Central Hospital and is added for its instructive value. Special stains were made in this study.

HISTORICAL ASPECTS

Seshachalam [5] suggests "that those who are concerned with the management of appendicitis should not be unmindful of the story of its development." From 50 B. C. to the present the appendix has manifested itself by signs and symptoms of pathology referable to the right lower quadrant of the abdomen. Leonardi da Vinci,[6] Carpi, Vesalius, and Fallopius, were early anatomists concerned with this anatomical entity, which was named the vermiform appendix by Vidius.

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* Augustana Hospital Series.

† St. Mary of Nazareth Hospital series.

Mestivier, in 1759, removed pus from the right lower quadrant of a patient subsequently autopsied; he found a large rusty pin in the vermiform appendix of this patient. Parkinson, Bright, and Addison, of Guy's Hospital, detailed signs, symptoms, and autopsy findings in appendicular disease.‡ Hancock, of Charing Cross Hospital, successfully operated upon a patient before "fluctuations" occurred. Sir Frederick Treves suggested removal of the appendix during a quiescent period. Charles Symonds, of Guy's Hospital, is given credit for the first successful interval appendectomy in 1885.

Reginald Heber Fitz [7] published his epochal report in 1886. He coined the term "appendicitis" rather than typhlitis and perityphlitis. According to McBurney,[8] Fitz "wrote vigorously and persistently and called attention to the size, shape and position of the appendix and its bearing on clinical conditions. From a vast experience gained at necropsy he maintained that the appendix was the cause of the trouble."

The modern period may be dated from the work of McBurney, in New York, who, in 1889, urged early surgery and paid much attention to the pathology. John B. Murphy,[9] of Chicago, crusaded for early surgery. His teachings included, "Get in quick, get out quicker, in spreading peritonitis; the earlier the surgical interference the better," and use of the Murphy continuous-drip isotonic sodium chloride infusion per rectum. Fowler,[10] of Brooklyn, contributed "Fowler's position" to the management of appendicitis and advocated surgery at the opportune moment. Ochsner,[11] in America, and Sherren, in London, 1904, championed the "starvation treatment."

PATHOGENESIS

Acute Appendicitis.—The factors involved are (1) obstruction of lumen, (2) retention of contents, (3) bacterial proliferation, (4) increase in intraluminal pressure, (5) ischemia, (6) bacterial invasion, and (7) tissue reaction.

In regard to the development of fulminating appendicitis, Wangensteen and Bowers,[12] Wangensteen and Dennis,[13] Van Zwalenburg,[14] and Aschoff,[15] working with the dog cecal appendage and on human appendices, and other writers in more recent publications,[4] have shown that there are two main factors as regards the exciting cause, obstruction and infection. Experimental evidence points to pressure-distention as the principal exciting factor, bacterial invasion of the injured wall playing a secondary role, albeit a specific diplococci has been implicated by Aschoff.

Subacute Appendicitis.—Most authors regard subacute appendicitis as one stage, either early or less virulent than the acute suppurative appendicitis. However, there are cases which are characterized pathologically by the excessive formation of granulation tissue microscopically identified as subacute granulation. Macroscopically the surgical specimen invariably presents an inflammatory mass somewhat well circumscribed, located usually at the site of previous inflammatory activity; it is soft and friable and bleeds easily when crushed with a forceps. Microscopically the stroma is composed of many budding capillaries surrounded by inflammatory cells. Cellular elements include monocytes, phagocytes of varying size and activity, some polymorphonuclear leucocytes, and a predominant number

‡ Cited by Seshachalam.[5]

of eosinophiles, and occasional plasma cells. These lesions may go on to complete resolution to become the appendicitis fugax of Kelly or the latent appendicitis of Bowers and of Fowler. The lesion may become activated and present signs and symptoms of a fulminating acute appendicitis.

Chronic Appendicitis.—Robert Moore [16] believes that both the clinical picture and pathological anatomy of chronic appendicitis are not yet to be delineated. Lockwood [17] believes true lymphoid hyperplasia is indeed a rarity, as most of the surgical specimens are those belonging to patients in that age group in which lymphoid hyperplasia is a normal incident. In the pathologically proved cases of chronic appendicitis two forms are recognized, the first form the commonest as a result of previous infection which had healed. The appendix becomes thickened, tortuous, and firm; the lumen remains mainly patent. Strictures may or may not be present. The second form includes the true chronic inflammatory infections. These follow infections with a specific agent, tuberculosis, actinomycosis, typhoid fever, amebiasis, blastomycosis, systemic sporotrichosis, coccidiomycosis. Appendectomy performed during the prodromal stage of measles reveals a lesion similar to that seen in the subepithelial tissue of the tonsil. Large multinucleated giant cells containing from 6 to 20 closely packed nuclei and an associated infiltration with lymphocytes and monocytes (Wharton) are characteristic.

PRESENT STUDY

In the 377 cases reviewed in this study, 302 appendectomies were performed for suspected appendiceal pathology. Appendectomies were done in 75 other cases in which the abdomen was opened without a preoperative diagnosis of appendiceal disease, as shown in the following listing:

Total no. of cases		377
Appendectomy: primary diagnosis "appendicitis"		302
Appendectomy with other surgery (*en passant*)		75
Pelvic surgery	45	
Cholecystectomy	19	
Celiotomy	11	
Gastric resection	2	
Ruptured peptic ulcer	2	
Hemicolectomy, right	2	
Inguinal herniorrhaphy	2	
Heineke-Mikulicz	1	
Umbilical hernia	1	
Intestinal obstruction	1	

In 200 cases of appendectomy performed with a diagnosis of appendiceal pathology, pain, nausea, vomiting, constipation, diarrhea, and distention occurred in the above sequence. Proportionately, with pain given a value of 100, nausea as a second symptom equalled 60; vomiting as a third symptom, 50; constipation as a fourth symptom, 20; diarrhea a fifth symptom, 12, and distention, the final symptom, 6. It became evident that a consideration of pain, nausea, and vomiting occurring in sequence became very important in the taking of the history, in the interpretation of the physical findings pertinent to the right lower quadrant, and

in the evaluation of the laboratory data. The above symptoms coupled with local findings were strong indications for surgical intervention and must be weighed against any existing contraindication.

Diagnosis.—Table 1 demonstrates the preoperative diagnosis of each group and the breakdown of these diagnoses as they appeared postoperatively. The distribution of the postoperative appendiceal pathology is not intended to show the incidence of that pathology. The 71% correct preoperative diagnosis compares favorably with

TABLE 1.—*Relation Between Preoperative and Pathologic Diagnoses*

Preoperative Diagnosis	Pathologic Diagnosis*					
	I	II	III	IV	Normal	Unusual†
I. Acute appendicitis (119)	66	20	12	9	12	..
II. Suppurative or gangrenous (31)	7	19	5	..	..	..
III. Subacute appendicitis (37)	8	2	19	..	8	..
IV. Chronic appendicitis (38)	..	..	18	19	1	..
V. Interval appendectomy‡ (77)	3	..	2	8	64	..
En passant§ (75)	..	..	..	32	22	21
Total cases (377)	84	41	56	68	107	21

* Roman numerals at the top of the columns represent diagnoses designated with numerals in the "Preoperative Diagnosis" column.
† Unusual lesions having symptoms of inflammatory changes are included in pertinent pathologic column.
‡ Interval appendectomies; surgery in quiescent stage.
§ En passant group denotes appendices removed secondary to other intra-abdominal pathology for which celiotomy was indicated.

TABLE 2.—*Unusual Lesions in the Appendix**

Lesion Totals	Indications for Surgery				
	Hernia	Laparotomy	Gall-Bladder Path.	Pelvic Path.	G. I. Path.
Subacute granuloma (5)	..	..	..	2	..
Carcinoid (9)	..	..	1	5	..
Carcinoma (1)	..	..	..	1	..
Diverticula (5)	..	..	..	1	2
P. arteritis nodosa (1)	..	..	..	1	..
Lymphangioma (1)	..	..	1	..	..
Measles (1)	..	..	..	..	..
Tuberculosis (1)	..	..	..	1	..
Endometriosis (1)	..	..	..	1	..
Mucosal hyperplasia (3)	1	..	..	1	..
Mucocele (11)	..	..	3	1	..
Calcific degeneration (2)	..	..	..	1	..
Miliary abscesses (1)	..	..	..	..	..
Enterobius vermicularis (5)	..	..	..	2	..
Chronic appendicitis (68)	1	4	10	2	12

* This Table shows total number and distribution of lesions; chronic appendiceal lesions are added.

that of other clinics. The number of appendices with no evidence postoperatively of acute infection also compares with Aschoff's figures. Twenty per cent of the subacute appendicitis cases present normal appendices at surgery, whereas the interval appendectomy in this series precludes, almost, a postoperative diagnosis of "appendiceal tissue."

It is to be noted that while chronic appendicitis was diagnosed preoperatively or at operation 38 times, in 19 of these cases the diagnosis was made by the hospital pathologists. The highest incidence of these lesions occurred in conjunction with chronic disease in female pelves. Gall-bladder disease (chronic cholelithiasis)

and peptic ulcer increased the incidence of this entity. Chronic periappendicitis, chronic nonspecific granuloma, and, rarely, active chronic appendicitis secondary to appendiceal disease (acute?) made up this total preoperatively. Only one case proved to be uninvolved pathologically, whereas 18 cases had evidence of subsiding inflammation in fibrosed or obliterated appendices. Sources of error included fibrosed, kinked, obliterated appendices with or without strictures and peritoneal bands misinterpreted as adhesions. Eight appendices removed at interval operation and 32 removed *en passant* were also chronically inflamed but presented no symptoms of primary appendiceal disease.

Pathology Incidence.—In this study there were 84 cases with postoperative pathologic diagnosis of acute appendicitis, 41 cases of suppurative or gangrenous appendicitis, 56 cases of subacute appendicitis, 68 cases of chronic appendicitis, 1 miliary abscess of the appendex, 1 case of measles appendicitis, 5 subacute granuloma, and others, totaling 47 cases, classified as unusual lesions (Table 2). Of these lesions 25 were removed when the abdomen was opened for other causes.

Of the nine carcinoid tumors of the appendix, six were found *en passant*, one during cholecystectomy and five in operations on the female pelves. One carcinoma of the appendix was found during the course of pelvic surgery, as was one case suspicious of polyarteritis nodosa of the appendix. Tuberculosis (tuberculosis of the tubes), endometriosis, mucocele, mucosal hyperplasia, calcific degeneration, and two worm infestations of the appendix were also found in the course of pelvic surgery. One carcinoid tumor of the appendix, one lesion suspicious of lymphangioma, and three mucoceles of the appendix were found at laparotomy for cholecystectomy. Thus 75 appendectomies performed *en passant* in the course of other operations yielded more than 30% of appendices which had unusual pathology. This percentage would reach very high proportions were the pathologically diagnosed chronic appendices (29) removed in similar circumstances to be added. Of the 75 appendices so removed, 51 showed pathology, 2 others had worm infestations.

Morbidity (Table 3).—In the 0 to 10-year age group there were 30 cases, of which 27 made an uneventful postoperative recovery, excluding one case that exhibited chills, nausea, and vomiting on the second hospital day, the day of surgery. Three cases had severe complications. Two of these had generalized peritonitis on admission and were treated conservatively by modified Ochsner-Sherren technique. These patients developed spiking fevers, increasing meteorism, pain in suprapubic regions with dysuria, and pyuria. One of these was operated on the 14th hospital day, the other on the 16th hospital day. Each had appendectomy and drainage of pelvic abscess as the initial surgical procedure. Pelvic abscesses redeveloped and eventually perforated through the rectum spontaneously. The third case was operated on the first hospital day. Two drains were inserted into abdomen and pelvis. This patient had a total hospitalization of 29 days, running a fever until the 23rd hospital day. Drains were removed on the seventh day. The abdomen continued to drain purulent exudate for 11 days more. The patient survived.

The factors increasing the morbidity of the appendicitis group included preoperative prolonged duration of illness before the patient sought medical aid; missed diagnosis; failure to recognize diarrhea; tarry stools; wound complications, such as infection and dehiscience, and pelvic abscesses, two of which ruptured spontaneously into the rectum and one that apparently resolved with no further complication.

Prolonged hospitalization included five cases that were hospitalized over 14 days, and four cases that were hospitalized over 21 days. Postoperative fever occurred in 21 cases and was the most prominent index to morbidity. Chills, vomiting, distention, and diarrhea occurred in pulmonary, peritoneal, and wound complications. Unexplained tachycardia and at least four days of fever usually existed before the wounds were found to be the site of complicating pathology.

In the 19 cases of appendectomy with gall-bladder surgery, fever, tachycardia, and right upper quadrant pain occurred in 4 cases; nausea, vomiting, and ileus were found to have the same incidence, 3 cases in 19. Clay stools associated with wound

TABLE 3.—*Morbidity*

	Appendectomy			En Passant		
	Acute	S/G*	U/N†	Gall-Bladder Surg. (19 Cases)	Pelvic Surg. (45 Cases)	G. I. Surg. (11 Cases)
Fever	11	8	2	4	6	4
Abdominal pain	1	6	4	4	5	2
Chills	3	4	2	2	2	1
Nausea, vomiting	2	2	1	3	3	2
Ileus	1	2	2	3	..	2
Cough	3	1	1	..	2	1
Chest pain	2	1	1	..	..	1
Diarrhea	2	2	1	..	..	2
Tarry stool	..	1	1	..	..	1
Clay stool	..	..	..	2	..	..
Pelvic abscess	..	3	..	..	..	1 (peptic ulcer)
Wound						
Infection	..	7	..	2	..	..
Dehiscence	1	..	1	..	..	..
Fistula	..	2	..	2	..	..
Pneumonia	1	1	1	..	..	..
Peritonitis	..	3	..	..	..	1 (peptic ulcer)
Shock	..	2	..	1	..	1 (peptic ulcer)
Hosp. over 21 days	..	2	1	2 (14 days)	1	..
	..	3	1	1	..	2
Deaths	..	2	1	..	..	1 (tumor cecum)

* S/G, suppurative or gangrenous.
† U/N, unusual or normal.

problems occurred twice, and shock occurred once. Two cases had hospitalization periods exceeding 14 days. One case had prolonged hospitalization of over 21 days because of jaundice and bile-draining abdominal fistula.

The postoperative course of the 45 cases in the group of pelvic surgery with appendectomy was generally uneventful. One case was hospitalized over 21 days. Fever, abdominal pain, nausea, vomiting, and cough were the symptoms of morbidity in this group.

Where appendectomy was done in connection with gastrointestinal surgery, fever was the predominant postoperative symptom heralding complication. Misdiagnosis (appendicitis for ruptured peptic ulcer) occurred on two occasions. One of these cases developed a pelvic abscess. Generalized peritonitis occurred after a right hemicolectomy.

SUMMARY

A clinicopathological study is presented of 377 appendices removed for primary disease or *en passant* during the course of other intra-abdominal surgery.

The clinical data included 189 cases from Augustana Hospital and 188 from St. Mary of Nazareth Hospital. One case, a carcinoid tumor of the cecum from the Illinois Central Hospital, was studied in conjunction with the other carcinoid lesions of the appendix appearing in this paper. Seventy-five appendectomies were performed *en passant*. Patients were followed preoperatively at surgery, or postoperatively whenever possible. Gross surgical specimens were examined whenever they were available. The microscopic sections were studied in each instance.

The literature is reviewed and serves as a basis for the presentation of such allied topics as the history of the appendix and its diseases, and pathogenesis and pathology of the appendix.

CONCLUSIONS

1. A painstaking history and a careful physical examination are indispensable in the diagnosis of acute appendicitis.

2. A correct diagnosis of acute appendicitis with subsequent appendectomy carries a low mortality and morbidity.

3. A high incidence of unusual pathology found in appendices removed *en passant* is noted.

4. Appendectomy performed in the course of other major surgical procedures within the abdomen of a patient prepared for such surgery did not appreciably add to the morbidity or mortality.

5. Postoperative fever, chills, pain, distention, vomiting, and unexplained tachycardia were indices to morbidity.

6. The over-all mortality in this series is 1.3%. The corrected mortality of 0.27% compares favorably with others.

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ATRESIA OF THE COMMON HEPATIC DUCT WITH SHUNT VIA AN ACCESSORY DUCT

Report of a Case

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NUMEROUS contributions to the surgical literature have been made regarding anomalies of the extrahepatic biliary system, with emphasis on potential surgical errors during so-called routine cholecystectomy.* Certain of the variations pose at first as normal cases, since the anomalous part lies just out of the restricted range of the immediate field of operation at the neck of the gall bladder. These are understandably misleading to the operator whose practice it is to attack the first structure that appears to be a bile duct joining that which appears to be the common duct and the first blood vessel that happens to lie in the usual position of the cystic artery at the neck of the gall bladder. Thus, the true nature of a devious main biliary tract is sometimes discovered only after ligation and division of one of its essential limbs. It is the purpose of this paper to reemphasize the need for thorough dissection of the gall-bladder pedicle before clamping its structures and to report an apparently unique combination of anomalies in this region.

Most of the writings on this subject deal with fairly common variations of position and number of the arteries and ducts.† The damage resulting from failure to appreciate the all too frequent occurrence of these commoner anomalies lies in injury to a main vessel or duct lying abnormally close to a structure involved in the operative procedure, i. e., a common hepatic duct paralleled by the cystic duct. Another error frequently mentioned is the division of an accessory bile duct without recognition of its presence.‡ Stolkind [30] mentions reports of this anomaly accredited to Pliny (23 to 79 A. D.) and even to Aristotle. He, however, states also that such descriptions were usually based on misconceptions of true anatomy, the result of inadequate dissection. Stolkind also quotes Schenkins (1564) and Diewerbroeck (1655), who apparently reported authentic anomalies of the region (double cystic ducts in each case). Since then, the literature is rife with reports and analyses of the usual types of anomalies § and with studies of atresia of the extrahepatic biliary tract.‖

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* References 5, 9, 11, 22, and 27.

† References 2, 3, 4, 7, 12, 18, 25, 26, and 31.

‡ References 10 and 19.

§ References 24, 25, 28, and 29.

‖ References 1, 6, 17, and 32.

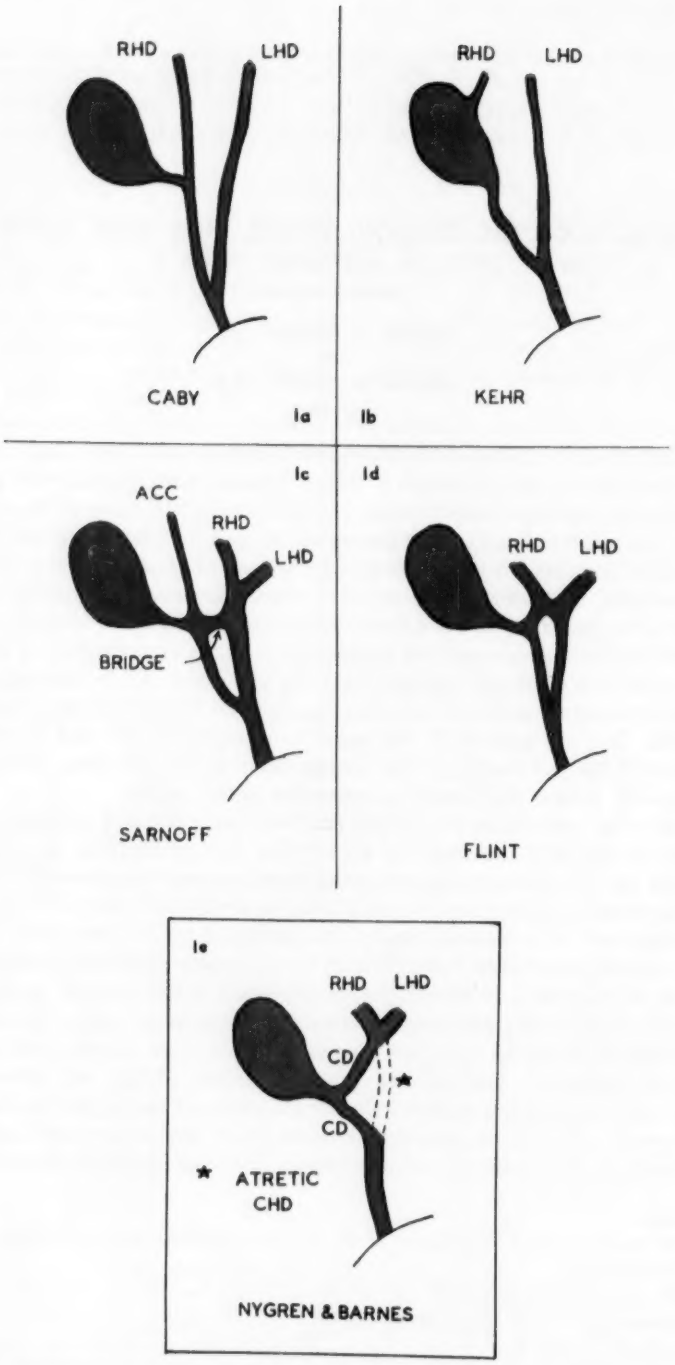

Fig. 1.—Anomalies of the extrahepatic biliary tree: *RHD*, right hepatic duct; *LHD*, left hepatic duct; *CHD*, common hepatic duct; *ACC*, accessory bile duct; *CD*, cystic duct.

Few articles, however, describe anomalies of such disposition that dividing, no matter how meticulously, what seems after "reasonable dissection" to be a normal cystic duct would result in a disaster or at best a chilling test of the surgeon's ingenuity and technical skill.¶ Caby [5] reported one such case (Fig. 1*a*). He was fortunate in being able, by ingenious surgical means, to extricate himself from the consequent difficulty. Kehr, in a discussion of Caby's presentation, described a similar case, but the operative result was not successful. Sarnoff [27] recognized the presence of an interesting anomaly before committing himself (Fig 1*c*). Kehr [15] reported another anomaly so treacherous as almost to deter one from ever clamping a duct before dissection of the entire gall bladder and pedicle (Fig. 1*b*).

These writers have mentioned the dearth of reports and discussions of these rare variations in the standard texts, atlases, and periodical reviews. The accessible literature in the commoner languages was reviewed, and few cases at all resembling the one here reported were found. The one most similar was reported by Kehr,[15] in

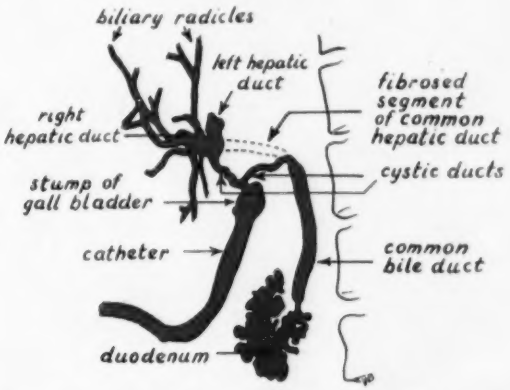

Fig. 2.—Drawing (from cholangiogram) showing anomaly of extrahepatic biliary tree with double cystic duct and location of fibrosed common hepatic duct.

1913, and in 1923 by Flint,[10] who encountered an accessory bile duct leading from the right hepatic duct, emptying into the cystic duct near the neck of the gall bladder (Fig. 1*d*). This finding was in accord with the statement often made that accessory bile ducts are almost invariably—some say always—accessory right hepatic ducts.[11] The anomalous duct described below, however, arose from the common hepatic duct just below the junction of right and left hepatic ducts (Fig. 2). No report was found describing combinations of atresia of the essential ducts and existence of accessory ducts in the same patient, as occurred in the case presented.

REPORT OF CASE

A 52-year-old housewife (New York Hospital No. 659383), referred by Dr. Mack Lipkin, was admitted to the New York Hospital on June 16, 1953. For many years she had complained of excessive postprandial gaseous eructation with occasional heartburn, not aggravated by fatty foods. Her physician told her about 12 years prior to admission that she had disease of the gall bladder. There had never been fever, nausea, or emesis, but about seven years before

¶ References 13 and 20.

admission she had experienced one episode of severe sharp right upper quadrant pain radiating to the right scapula, lasting several hours. There was no history of jaundice, dark urine, or acholic stools and no complaint relative to the remainder of the gastrointestinal tract. A recent cholecystogram demonstrated cholelithiasis; an electrocardiogram and x-ray of the chest were normal. She had borne no children, and a hysterectomy had been performed 10 years prior to admission. Review of her systems was unremarkable.

She was small but well developed and apparently healthy. There was no icterus, and there were no other abnormalities except a well-healed scar in the left lower quadrant of the abdomen and a granular cystic cervix uteri.

Examinations of the blood cells and urine were normal. Cervical biopsy revealed chronic cervicitis, and cytological study of vaginal, cervical, and endocervical smears revealed no evidence of neoplasm.

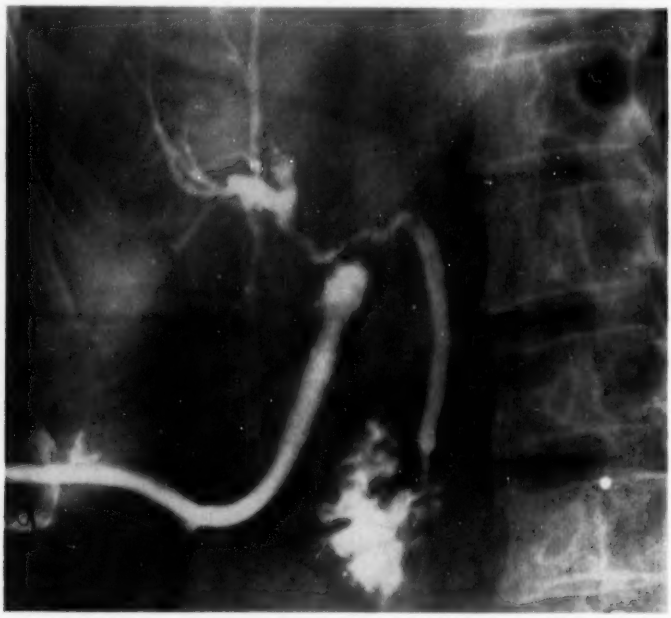

Fig. 3.—Cholangiogram showing Diodrast in biliary radicles, cystic ducts, remnant of gall bladder, common bile duct and duodenum, but absence of dye in the common hepatic duct.

Through an upper right rectus incision, the abdomen was thoroughly explored, including the entire small bowel. The uterus was absent, and several calcified left iliac lymph nodes were palpated. The liver appeared normal. Several small stones were present in the gall bladder, the wall of which was moderately thickened. Several fine adhesions connected the gall bladder and the distal stomach, duodenum, and omentum. The adhesions were lysed, and the peritoneum over the gall-bladder pedicle was incised and reflected. A cystic duct and an artery, normal in appearance and location, were immediately encountered. However, dissection up to the neck of the gall bladder disclosed a second duct that arose from the same level of the neck of the gall bladder as the first, running superiorly toward the porta hepatis, where it joined the common hepatic duct just distal to the junction of the right and left hepatic ducts (Figs. 1*e*, and 2).

Because of this peculiarity, the gall bladder was dissected from the fundus toward the pedicle. No other anomaly was found at this site. The cystic artery was divided between clamps and ligated. It was noted that, whereas the wall of accessory duct was translucent, pliable,

and distended with bile, which could be seen flowing back and forth within the lumen, the common hepatic duct, between its junction with the accessory duct and cystic duct, appeared opaque and more rigid, and no contents were visible. Attempts to aspirate bile from this segment were unsuccessful, and after the injection of a solution of 35% iodopyracet (Diodrast), first into the common bile duct and then into the accessory duct, there was no radiographic evidence of a lumen in the common hepatic duct. An incision 1 cm. in length was made in the wall of the latter duct. No lumen was found, and no bile emerged. With complete stenosis of the common hepatic duct, the biliary shunt furnished by the accessory duct and the cystic duct was the only passage through which bile could reach the duodenum.

The gall bladder was removed, leaving a segment approximately 1 cm. in length at the neck to which the two ducts were attached. An attempt was made to introduce a T-tube into the stump of the gall bladder with one segment of the cross limb of the tube in each duct. However, although the orifice of the accessory duct admitted one limb of the T-tube, the opening of the cystic duct could not be probed because of a fold of tissue at the ampulla, probably one of the valves of Heister. Another means of decompression of the biliary tract was therefore necessary.

The mucosa was stripped from the neck of the gall bladder, and the cuff of the remaining wall was closed over the end of a No. 14 French whistle-tipped rubber catheter, 000 interrupted nonabsorbable surgical (silk) sutures being used. The catheter was further anchored with 0000 chromic absorbable (gut) suture. Bile drained freely from the catheter. A single cigarette drain was placed at the foramen of Winslow and brought out through a lateral stab wound with the catheter. The peritoneum was closed with chromic gut, and the abdominal wall in layers, with silk sutures.

The patient's postoperative course was benign. The catheter drained little bile, and only a small amount of bile emerged along the cigarette drain. She remained free of jaundice. Determinations of total bilirubin on the 5th and 20th days after operation were 0.3 and 0.2 mg., respectively. The cigarette drain was removed on the fourth postoperative day. A cholangiogram was obtained on the 14th postoperative day. There was prompt unobstructive flow through the accessory duct into the hepatic radicles and through the cystic duct into the common duct, thence into the duodenum. The common hepatic duct was not visualized (Fig. 3). The catheter was removed two days later. She remained afebrile and was discharged on the 20th postoperative day with the wounds healed. She remains asymptomatic six months after operation.

COMMENT

Many embryologists believe that at an early stage of life developing hollow structures become occluded by an epithelial proliferation that in preparation for rapid growth and expansion lays down tissue so profusely that the ducts become solid structures, to be recanalized later as growth proceeds.# Failure to recanalize is the most widely accepted theory of etiology of congenital strictures of the biliary tree. The embryology of accessory ducts has not been so widely discussed. They are thought to be the result of heterotopic growth, not of any particular stimulus or design.[2] There is no evidence that biliary atresia acts as a stimulus to formation of shunts or accessory ducts. Atresia presumably occurs either too late in development to be compensated for by heterotopic duct growth, or, if present early, the secretion and flow of bile begin too late to influence the development of the ducts.[14] When atresia involves the main ducts, there usually is no way for bile to enter the intestine, short of surgical intervention.[16] In the case presented a fortunate freak of development (an accessory duct) enabled bile to bypass an atretic segment of the common hepatic duct, probably a unique combination among an already rare group of anomalies of the extrahepatic biliary tree. It cannot be stated with certainty whether the duct that connected the gall bladder with the proximal end of the com-

References 8, 14, 16, and 21.

mon hepatic duct was a second cystic duct or an accessory bile duct of the type described by Flint [10] (Fig. 1*d*). The thin-walled appearance and apparent absence of valves suggested that it was an accessory bile duct but its attachment to the common hepatic duct lent weight to the belief that it was a cystic duct. The terminology is less important than the fact that the occurrence of an anomalous duct in such a position as to provide a shunt about the atretic hepatic duct saved this patient either from an early death due to biliary cirrhosis or from a difficult operation, namely an attempt to anastomose the common hepatic duct at its origin with the gall bladder, common bile duct, or jejunum.[28]

Only after thorough dissection of the gall-bladder pedicle had been performed was there the least suspicion of anything unusual in the disposition of the extrahepatic biliary tree. A normal cystic duct was earlier seen in its usual position entering what certainly was the common bile duct. Yet, had this structure been clamped and ligated at this time, a disastrous surgical error would have been committed, the result being equivalent to transection of the common bile duct.

Dissection of the gall bladder from the fundus toward the cystic duct, rather than immediate division of the cystic duct and removal of the gall bladder in the reverse direction, has been advocated by many surgeons. By this method abnormalities may be recognized before the surgeon has committed himself and the unfortunate results that follow transection of unrecognized vital structures about the gall bladder may be averted.

SUMMARY AND CONCLUSIONS

A unique case is presented in which atresia of the common hepatic duct was associated with an anomalous duct connecting the proximal end of the common hepatic duct with the neck of the gall bladder, providing a shunt for the passage of bile to the intestine.

During cholecystectomy, prior to division of any vessel or duct, complete dissection of the gall bladder, particularly in the region of the pedicle, will insure recognition of anomalies and so prevent transection of essential structures.

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EVALUATION OF NEOMYCIN-PHTHALYLSULFATHIAZOLE IN PREPARATION OF THE LARGE BOWEL FOR SURGERY

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THE NEED for an effective intestinal antiseptic was evidenced as early as 1874, by Billroth [1] in his work on intestinal antisepsis. But no significant advance in this direction occurred until Poth [2] introduced succinylsulfathiazole as an adjunct in the preparation of the large bowel for surgery. Despite the use of new antibiotics within the past five years, including streptomycin, chlortetracycline (Aureomycin), chloramphenicol, and oxytetracycline (Terramycin), no single agent has proved completely satisfactory for all cases. A variety of unfavorable reactions, including diarrhea, proctitis, pruritus ani, pseudomembranous colitis with fatalities,* and outgrowth of resistant organisms has been noted with the use of certain antibiotics as intestinal antiseptics.

For effective use in the preparation of the colon or rectum, Poth [5] has set forth the properties of an ideal intestinal antiseptic. It is unlikely that any single antimicrobial drug will be found which fulfills all of these criteria, but it is possible that some combination of drugs may approach the ideal. Satisfactory results over a five-year period with the use of streptomycin and phthalylsulfathiazole (Sulfathalidine) as adjuncts in bowel surgery have been obtained, with relative freedom from untoward reactions such as overgrowth of fungi and pseudomembranous colitis reported with other antibiotics, by one of us (H.E.B.).

This method of intestinal antisepsis was initially reported by Rowe, Spaulding, Madajewski, and Bacon [6] in 1948 and later [7] described in more detail.

In 1950 Poth [8] recommended neomycin as an intestinal antiseptic superior to those previously available. Because of the frequent appearance of resistant Aerobacter organisms, however, he subsequently [9] adopted a combination of neomycin and phthalylsulfathiazole. The excellent results reported by him [10] prompted the present study.

PROCEDURE

Clinical confirmation of the rapid bowel preparation was made in a series of patients undergoing surgery of the colon and rectum, with the administration of 10 gm. of neomycin and 15 gm. of phthalylsulfathiazole in divided doses over a 24-hour period, immediately prior to

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* References 3 and 4.

surgery. The agents were orally administered in the form of a single combination tablet (neomycin-phthalylsulfathiazole [Neothalidine†]). The following dosage schedule was employed: 5 tablets, each containing 200 mg. of neomycin and 300 mg. of phthalylsulfathiazole, were given orally, at hourly intervals for four hours; thereafter 5 tablets were given orally every four hours until a total of 50 tablets had been administered, making a total of 10 gm. of neomycin and 15 gm. of phthalylsulfathiazole given in the 24-hour period immediately preceding large bowel surgery. The last dose was given approximately six hours before making the incision.

In addition to the combination tablets, mechanical cleansing of the bowel was achieved by the oral administration of 60 cc. of castor oil given 36 hours prior to surgery or in divided doses if partial obstruction was present. Cleansing enemas of isotonic sodium chloride solution were given daily for three days prior to surgery and on the morning of the surgery. No solid foods were permitted the day preceding the surgery, liquid feedings being substituted. The selection of patients for this regimen of castor oil, enemas, and neomycin-phthalylsulfathiazole preparation precluded any patient in whom obstruction was a permanent factor requiring management different from primary resection without complementary colostomy. All patients in this study were treated in the Philadelphia area, during the winter and early spring, receiving a high-protein, high-carbohydrate, low-residue diet with protein supplement. No other intestinal antiseptics were used.

Data on Neomycin-Phthalylsulfathiazole-Castor Oil Effect on Microbial Flora of Bowel in Sixteen Patients

Average Colony Counts (In Thousands)

	Preliminary Stool	Operative Fecal Specimen	Final Postoperative Stool
"Total" aerobes	1,000,000	400	2,000,000
"Total" anaerobes	5,000,000	1,000	40,000
Coliform bacilli	6,500	0*	80
Spores	8	0†	10
Enterococci	60	0†	2.5
Yeasts	31	0.8	15

* Less than 5,000 per gram of wet fecal material, the smallest number detectable by test method.
† Less than 100 per gram.

The patients receiving this preparation have been divided into two groups. Group I consists of those patients having comprehensive microbiologic study of all stool specimens preoperatively, at the time of surgery, and postoperatively, in order to determine precisely the effect of neomycin-phthalylsulfathiazole upon the microbial flora of the large intestine. Group II consists of those patients who received neomycin-phthalylsulfathiazole after the completion of the cases in Group I. Thus, Group II patients were studied for clinical results and toxicity, while Group I patients were studied mainly for the microbiologic effect.

The 16 patients in Group I were observed for an average period of 11.7 days, and an average of nine specimens were examined from each patient. These included preliminary control stools, stools following castor oil, stools following neomycin-phthalylsulfathiazole tablets, fecal specimen taken at the time of surgery, and postoperative stools. The time at which the first postoperative stools were obtainable ranged from two to seven days after the operation.

Microbiologic examination consisted of "total" aerobic and anaerobic colony counts and of separate cultures on special media for coliform bacilli, micrococci (staphylococci), streptococci, yeasts, and spores. The predominating types of organisms were selected from the 20 culture plates routinely used and tested for neomycin susceptibility. In addition, the majority of stool specimens were assayed for the neomycin content.

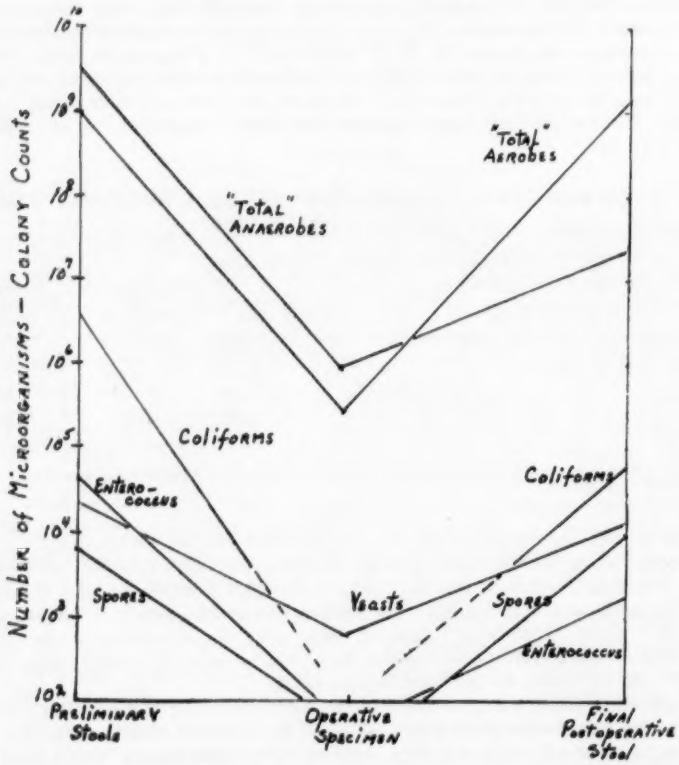
RESULTS

Group I.—A summary of the culture results appears in the Table. The number of coliform bacilli decreased rapidly, beginning with the first enema. In no

† The material used in this study was supplied by Sharp & Dohme, Inc., Philadelphia.

instance were coliforms detectable in the operative specimens; this means that the average number decreased from 6,500,000 in the preliminary specimens to less than 5,000 per gram of wet feces, which is the smallest number detectable by the culture method employed. In five cases these organisms were even absent from the post-operative specimen.

Spores and streptococci are normally present in relatively small numbers, 8,000 to 60,000 per gram. Therefore it is not surprising that these types were usually absent in the operative specimens. Because it is unlikely that neomycin-



Average colony counts with 16 patients receiving neomycin-phthalylsulfathiazole and castor oil in preparation for surgery.

phthalylsulfathiazole can bring about rapid destruction of spores, it is assumed that a disappearance of these resistant forms was the result of mechanical cleansing. The decrease in the number of streptococci can also be explained by the mechanical action of the castor oil and enemas.

On the other hand, yeast forms, which were present in all but two of the preliminary stools in approximately the same numbers as the streptococci, persisted in over half the cases. This fact suggests that the desirable results already mentioned with the coliforms were due to a combined purgation and drug effect.

There was a marked variation in the total number of micro-organisms detectable at the time of operation. Since no single culture medium is capable of growing all of the numerous varieties of microbes in feces, the figures representing the number of colonies present on duplicate aerobic and anaerobic trypticase soy agar are referred to as "total" counts. In some cases the decrease was insignificant; in others the effect was so remarkable that only 1 organism in 6,000,000 persisted. The average figures for the 16 patients under study (Table) show that the "total" aerobes decreased by 99.96% and the "total" anaerobes by 99.98%. These results are presented graphically (Chart).

Group II.—This group included 39 patients, which, combined with those of Group I makes a total of 55 patients having neomycin-phthalylsulfathiazole preparation for large bowel surgery in whom no significant toxicity was encountered. There were five instances of gross peritoneal contamination inadvertently occurring at the time of open anastomosis or secondary to inadvertent escape of fecal content from the bowel. One of these patients died on the 11th postoperative day, as result of retroperitoneal sepsis; Proteus organisms were recovered from an abscess culture. No other mortality occurred on the basis of sepsis.

COMMENT

The results reported here agree with those of Poth ‡ to the extent that preparation of the bowel with large doses of neomycin and phthalylsulfathiazole combined with castor oil and enemas is highly effective. Coliform bacilli are absent at the time of operation, as in most instances also are spores and enterococci. In further agreement with that investigator, yeasts persist throughout such a regimen.

On the other hand, the present report fails to demonstrate the sterility state reported by Poth.[10] Although completely negative cultures were obtained from one operative specimen, "total" counts in the range of 100,000 to 1,000,000 per gram were generally obtained. Because of the large doses of neomycin-phthalylsulfathiazole used in the bowel preparation described in this report, heavy concentrations of the drugs may be present in fecal specimens. These yielded assay values as high as 130 mg. per gram of feces, which are great enough to produce a bacteriostatic effect in the cultures made from low dilutions. When the proper technique was employed, however, it could be demonstrated that such specimens still contained many viable micro-organisms. Thus, we have been unable to demonstrate actual sterilization of feces.

The best results were obtained among the patients in Group I, in whom the response to castor oil was most marked. This observation emphasizes the comment made by Poth [10] that the length of time required for the preoperative preparation of the bowel is determined by the degree of mechanical cleansing obtained. Furthermore, it raises the question as to whether the mechanical effect is not more important than the antimicrobial action of neomycin-phthalylsulfathiazole. Regardless of the actual roles played by the two agencies, a combination of castor oil, enemas, low-residue diet, and neomycin-phthalylsulfathiazole causes a marked reduction of the microbial population of the feces, provided a satisfactory emptying of the bowel occurs.

‡ References 9 and 10.

It must be emphasized that the microbial counts of the operative specimens are misleading. Sometimes a small bolus of fecal material was all that could be found at the time of resection, but this small specimen, when cultured, would often yield a comparatively high microbial count, in terms of the numbers of microorganisms per gram of feces. Although the figures thus obtained appear in the Table, it is apparent that they fail to reflect the fact that the bowel was practically empty at the time and actually contained comparatively few organisms.

Neomycin administered intramuscularly has been shown to cause both ototoxicity and nephrotoxicity, but when administered in large oral doses, up to 10 gm. a day as was done in this study, the amount absorbed from the intestine is negligible. The range of the urinary recoveries following the administration of 10 gm. of neomycin is usually less than 1% and has only occassionally been observed to be as high as 5% of the administered dose. Accordingly, neomycin administered orally is free of toxicity assigned to it following parenteral administration and by reason of nonabsorption remains in the gastrointestinal tract where it exerts a prolonged antimicrobial effect, an important property in the crucial period of relative ileus following bowel resection.

SUMMARY

A program of preoperative bowel preparation that combines mechanical cleansing with large doses of neomycin and phthalylsulfathiazole for 24 hours was carried out with 55 patients.

Extensive microbiologic studies were made in 16 cases. In confirmation of Poth's findings, coliform bacilli, spores, and strepococci were usually absent from the operative specimens.

Wide variation occurred, however, in the "total" microbial counts, depending upon the extent of the mechanical cleansing. The average reduction was 99.96% for aerobes and 99.98% for anaerobes. An occasional instance of apparent sterility was attributed to a false bactericidal effect produced by high concentrations of drugs carried over to the culture plates.

No significant toxicity was encountered after the oral administration of neomycin-phthalylsulfathiazole as described.

The regimen first suggested by Poth and followed in this study appears to be an effective method for bowel preparation. Because the drugs are administered for only 24 hours prior to surgery, there is little opportunity for overgrowth with yeasts and other resistant organisms before the time of operation.

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ANATOMY OF THE DEEP VENOUS SYSTEM OF THE POPLITEAL FOSSA AND LOWER LEG

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MIAMI BEACH, FLA.

THE DEEP venous system of the popliteal fossa and lower leg is a neglected field of anatomy that is acquiring increasing importance. Chronic venous insufficiency may be caused by disturbances in these veins, and our attention has been focused on them in order that we might diagnose and treat this condition. One of the chief means of evaluating these vessels is by roentgenography, by the so-called phlebogram. However, interpretation of results in this test has been difficult, in a great share due to lack of understanding of the vein branches visualized. Somewhat similarly a lack of accurate anatomic orientation may account for differences in results noted when the popliteal vein has been ligated as a therapeutic measure in instances of "phlebitis."

In perusing standard texts of anatomy,* it is remarkable how little attention has been devoted to the deep veins of the lower leg. Generally, though, there is agreement that the popliteal vein is formed by union of the posterior and anterior tibial veins and that the peroneal vein is a branching of the posterior tibial vein. Scant attention has been paid to variations occurring in this pattern or to the lesser muscular branches.

With these thoughts in mind, we have attempted to examine this system in more detail and accordingly have dissected 57 legs in 57 routine autopsied cases. The popliteal fossa and deep compartments of the leg were exposed posteriorly by a skin incision from midthigh to the ankle and muscles either separated or cut to visualize the anatomy. In addition, branches of the anterior tibial were followed by splitting the interosseous membrane downward from the point of anterior projection of this vein between the tibia and fibula. Intramuscular branchings were examined by multiple sectioning of the soleus and gastrocnemius muscles.

ANATOMIC FINDINGS

Four general patterns were found for the anatomy of the popliteal vein and its principal branchings (Fig. 1).

I. In this type, found in 20 instances, the popliteal vein was lengthy and single and received its anterior tibial and posterior tibial branches immediately below the popliteus muscle at the level of the bifurcation of the popliteal artery. The peroneal vein likewise joined the posterior tibial at or slightly below this point.

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* References 1 to 6.

II. In nine cases the anterior tibial vein after piercing the interosseous membrane as in Type I passed over the lower margin of the popliteus muscle to enter the posterior compartment of the leg. It then journeyed 2 to 4 cm. cranial before joining the popliteal vein. The latter vein was formed by the union of the peroneal and the posterior tibial veins. In this type, occasional accessory small anterior tibial veins entered the peroneal or posterior tibial veins below the main anterior tibial branch.

III. In this type, found 22 times, the anterior tibial vein joined with either the peroneal vein or the posterior tibial to form a "popliteal" channel which as it coursed upward was joined by the remaining posterior tibial or peroneal vein. This latter junction occurred as high as the knee joint level. Usually the anterior tibial united with the posterior tibial as visualized in Fig. 1.

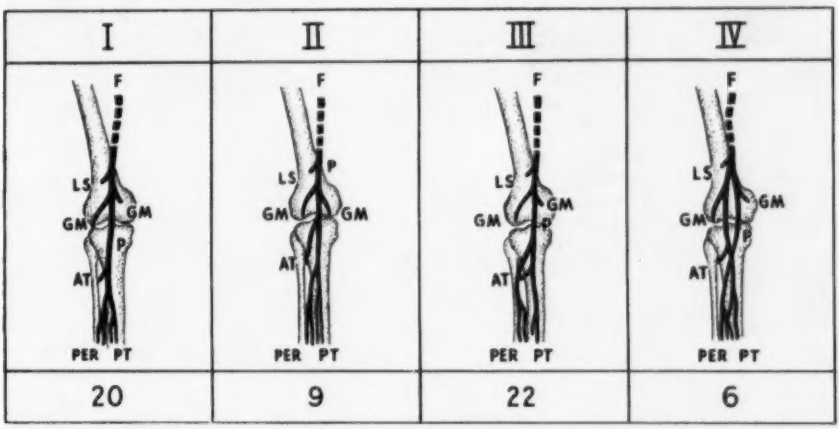

Fig. 1.—Incidence of main anatomical types of deep venous system of the popliteal fossa and lower leg in 57 autopsy dissections. *LS*, lesser saphenous vein; *GM*, gastrocnemius muscle vein; *AT*, anterior tibial vein; *PER*, peroneal vein; *PT*, posterior tibial vein; *P*, popliteal vein; *F*, superficial femoral vein.

IV. Here the popliteal vein was bifid either throughout or in its more distal portion. This was observed in six instances.

Generally the lesser saphenous vein was found to terminate a few centimeters above the union of the gastrocnemius branches with the popliteal vein. However, in many cases the termination was into the deep femoral branchings or superficial veins at the apex of the popliteal fossa. Venous branches from the gastrocnemius muscle in all instances were two in number, one branch passing to each muscle head at the level of the condyles of the femur. The soleus muscle venous drainage was chiefly into a major branch of the peroneal veins. Smaller veins emptied into both posterior tibial and peroneal branchings. Large venous channels in many instances were observed in the soleus muscle and less commonly in the gastrocnemius muscle. The nature of these channels is now under study. Geniculate branchings to the area of the knee joint were usually small and variable at the midportion of the popliteal vein. The posterior tibial and peroneal veins were frequently doubled,

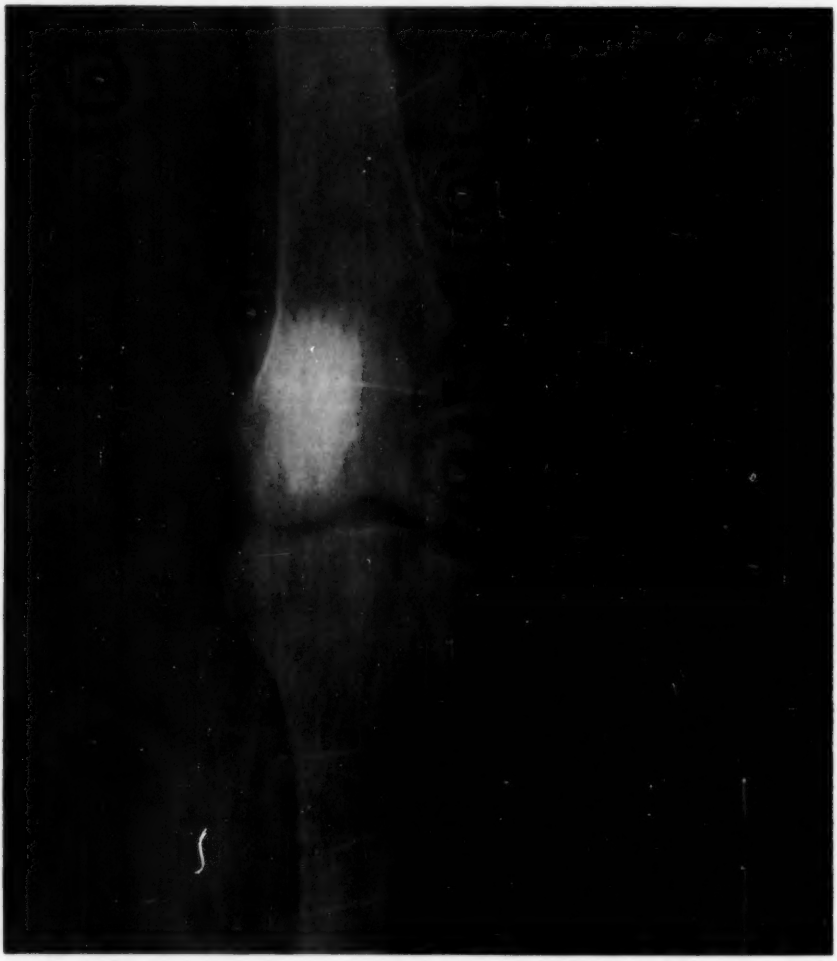

Fig. 2.—Location of anatomic landmarks and main deep venous branchings of popliteal fossa and lower leg as visualized roentgenographically after autopsy leg dissection. Pins placed as follows: (*A*) upper border of popliteal fossa; (*B*) gastrocnemius muscle vein branches; (*C*) lower border of popliteal fossa; (*D*) anterior tibial vein branchings; (*E*) bifurcation of posterior tibial vein (or lower popliteal vein) into terminal posterior tibial and peroneal branches, and (*F*) level where large vein channels were noted in soleus muscle.

though not at their termination. Collateral venous pathways were for the most part not well developed between the gastrocnemius and soleus muscles, nor between these muscles and the superficial areas of the limb.

The diameter of the vessels varied considerably from posterior tibial and peroneal branches of only a few millimeters diameter with a popliteal vein of 0.5 cm. diameter to posterior tibial and peroneal vessels of 1.0 cm. diameter with a popliteal vein of 1.5 cm.

With respect to surface and roentgenographic correlation, it was found that the exposed popliteal fossa extended rostrally approximately 11 cm. from a point a few centimeters above the level of the fibular head. This lower level of the fossa roughly corresponded to the lower popliteal skin fold as found on the surface of the leg. The gastrocnemius muscle venous branchings were noted at a level about 4 cm. above the fibular head. The anterior tibial entered the popliteal vein at a level ranging from a few centimeters below the gastrocnemius muscle branches to 6 cm. below the fibular head. The large channels within the gastrocnemius lay at about 6 cm. below the fibular head, while the similar channels in the soleus muscle were observed at about 9 cm. below the head. In one case a roentgenographic visualization of some of these phlebographically important points was obtained by placing pins at the level of the exposed vessel branchings and other landmarks (Fig. 2).

In general, the popliteal artery and vein in the popliteal fossa were loosely encompassed by muscles and showed collateral outlets to the surface. However, beneath the soleus muscle all vessels were covered by heavy musculature except medially at the lower posterior tibial area. From a venous standpoint the leg thus possesses a loosely bound popliteal fossa compartment within which lies a major share of the popliteal vein with its lesser saphenous, geniculate, and gastrocnemius muscle branches, and a tightly bound compartment beneath the soleus muscle housing the lower popliteal vein and its multivalved, smaller anterior and posterior tibial and peroneal branches. The anterior tibial vein passing forward through the interosseous membrane drains the anterolateral muscle groups and the anterior surface of the foot and is closely encompassed by muscle masses to the ankle.

COMMENT

From our appraisal of the anatomy of the lower limb, it would appear that the generally rigid concept of the origin of the popliteal vein from a union of the anterior and posterior tibial veins does not always hold. Likewise, the peroneal vein frequently does not terminate in the posterior tibial, and the anterior tibial too demonstrates considerable variation in its termination. Most consistent is the fact that a structure, which could best be called the popliteal vein throughout its entirety, usually extends from the level of the bifurcation of the popliteal artery to the adductor magnus muscle. The fact that according to most attempts at classification the lower part of this vessel might in some instances be called the upper part of the posterior tibial or peroneal veins is to us not as important as the point that in size and structure this segment is akin to the popliteal vein. Thus, this segment might best be termed the "lower popliteal" vein.

The deep venous system posteriorly from the level of the lower margin of the popliteus muscle distally appears to be related to return of blood mostly from the soleus muscle, ankle region, and foot, and not from the surface tissues of the calf.

Branches of the deep venous system in the popliteal fossa, on the other hand, drain the upper third to half of the deep tissues of lower leg and the surface tissues of the posterior aspect of the calf. Anteriorly and laterally the anterior tibial veins appear to play an important role in drainage of associated muscle groups and the surface tissues.

In chronic venous insufficiency these anatomical relations are of importance, particularly with respect to the manner of transmission of the high, unrelieved, venous pressures to the surface of the lower leg. In the popliteal fossa, because of the lack of muscle support and the presence of many pathways to the subcutis, transmission will readily occur to the superficial tissues. In the more distally placed deep venous elements below the popliteal fossa transmission of pressures will not be as likely because of the heavy supporting musculature.

Where ligature or resection of the popliteal vein or its branches is contemplated, it would be advisable to examine the anatomic situation carefully at the time of surgery. The type of usual vessel pathways and variations must be looked for. Collateral channels must be assessed. Access to the popliteal vein would be easiest in the lower popliteal fossa and most difficult in the "lower popliteal" vein segment. Separation of the popliteal vein from its associated anterior lying artery is difficult in most instances, particularly in the lower reaches of the vein. Branchings in this lower area are also difficult to separate from their accompanying arteries.

SUMMARY AND CONCLUSIONS

Fifty-seven lower leg and popliteal fossa dissections have been performed in 57 routine autopsies and the pattern of the deep venous system noted. Four general anatomic types have been observed for the popliteal vein and its branches. It was also noted that vessels in the popliteal fossa were loosely confined by muscles, while veins in the lower leg, except at the ankle, were closely confined. Anastomoses between the superficial and deep venous systems were frequent in the popliteal fossa and infrequent distally.

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SOLITARY DIVERTICULITIS OF THE CECUM

Report of Case

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DIVERTICULITIS of the colon is a fairly common condition, but the majority of cases of diverticulitis involve the sigmoid colon. Mayo [1] in 1950 reviewed 202 cases of diverticulitis of the colon, and, of these, 198 were in the sigmoid and only 2 were in the cecum.

In the case which is being reported there was a solitary diverticulum of the cecum. Of the previously reported cases of diverticulitis of the cecum, most were solitary diverticula. Anderson [2] in 1947 published a complete review of the literature, and, including 8 cases which he reported, he was able to find 99 cases of acute diverticulitis of the cecum. In this series the average age of the patients was 39.1 years, as compared with the average age of 54 years for patients with all types of diverticulitis as reported by Mayo. The sex incidence was about equal between males and females.

Ahlquist and Kepl [3] reported a case in May, 1952, and collected 103 cases.

The symptoms are frequently confused with acute appendicitis and in cases in which previous appendectomy has been done they may be confused with carcinoma of the cecum. At operation the findings may still be confused with carcinoma of the cecum because of the large inflammatory mass present.

Some attempt has been made to classify the diverticula into congenital and acquired types,[4] depending on whether the muscular wall is intact. With use of such a criterion our case would fall into the acquired type.

Herbut [5] states that diverticula of the cecum are located on the anterolateral or posteromedial borders, whereas in the colon they are almost always between the mesocolic and antimesenteric taeniae.

REPORT OF A CASE

O. C., a 32-year-old, white, married man, was admitted to the hospital on Feb. 12, 1953, complaining of severe right lower quadrant pain of 72 hours' duration. Seventy-two hours prior to admission the patient had noticed some constipation and had taken magnesia magma for relief. He awakened with mild cramping paraumbilical pain and had a normal bowel movement, with relief. The next day the pain shifted to the right lower quadrant and became constant and progressively severer.

Past history was noncontributory, with the exception of appendectomy performed for acute appendicitis in 1943. After the appendectomy the patient's postoperative course was complicated by an ileus which lasted approximately 10 days.

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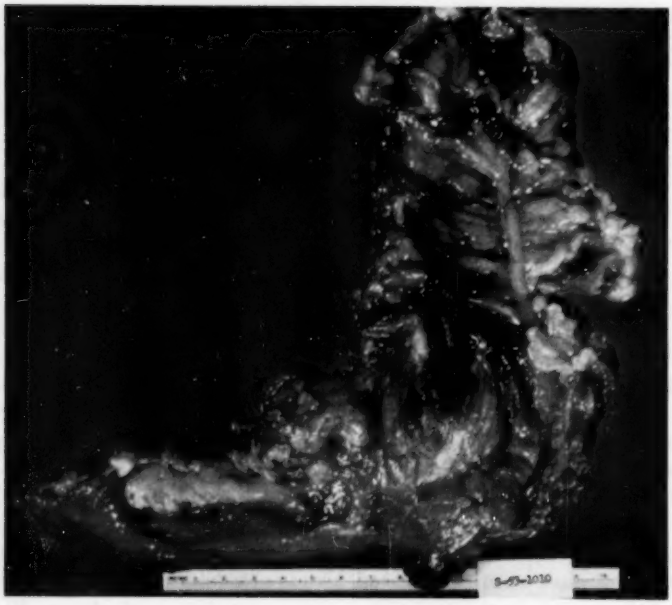

Fig. 1.—Mass of striated muscle and connective tissue adherent to posterior wall of cecum.

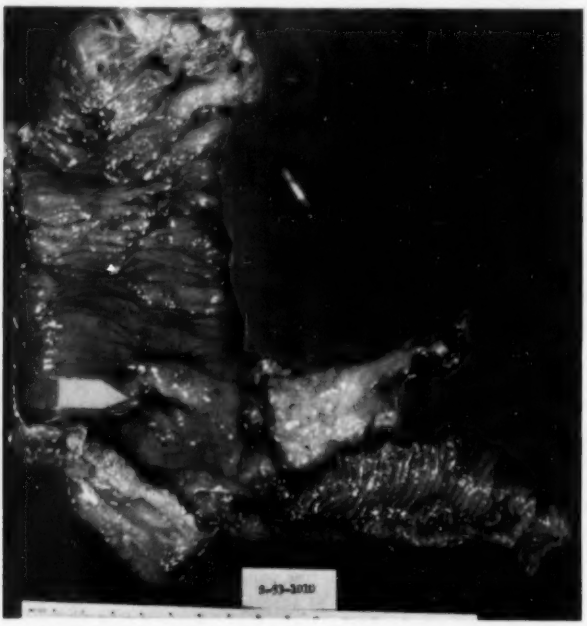

Fig. 2.—Ulcer crater on posteromedial wall of cecum is indicated by arrow.

Examination revealed a moderately toxic, thin, white man, with a temperature of 100.1 F. and a pulse rate of 100, who appeared acutely ill. Examination revealed marked tenderness lateral to the previous McBurney incision scar in the right lower quadrant. There was marked rebound tenderness in the right upper quadrant. There was rigidity and guarding in the right side of the abdomen, but the rest of the abdomen was soft. There was a bulge visible beneath the McBurney scar and audible bowel sounds over this area. On rectal examination some tenderness was revealed high in the right side and there was no stool in the ampulla.

Laboratory examination revealed WBC of 17,200, with 75% segmented forms. The urine was clear, with occasional white blood cells in the centrifuged specimen. An abdominal x-ray examination was ordered and carried out immediately. Supine, erect, and lateral decubitus films revealed no evidence of ileus or obstruction and no free air under the diaphragm.

Preoperative diagnosis: Possible strangulated small bowel or cecal abscess and exploratory laparotomy advised.

Immediate exploratory laparotomy was undertaken, and the abdomen was opened through a right rectus incision of the muscle-splitting type extending from just above the pubis to 2 in.

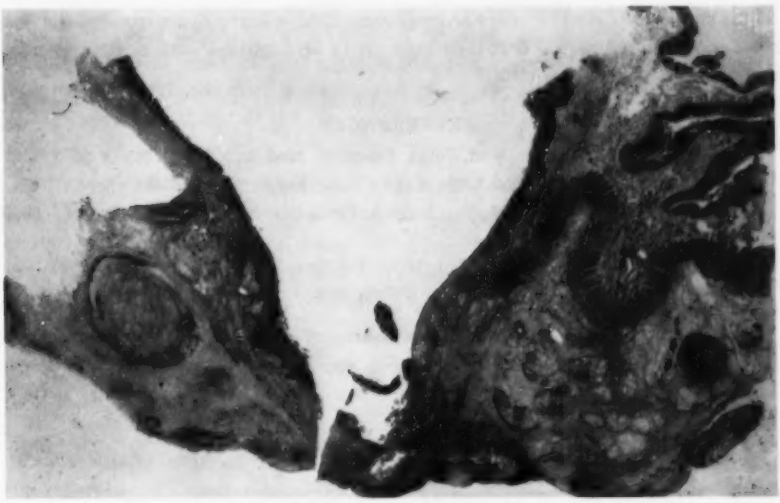

Fig. 3.—Photomicrograph taken at ×9 magnification shows penetration of ulcer through muscular wall into adjacent connective tissue.

(5 cm.) above the umbilicus. Examination revealed moderate edema and adhesions between the cecum and the lateral parietal wall. These were freed by combined blunt and sharp dissection and there was marked edema in these tissues. When this area was freed, an inflamed mass of a plaque-like nature about 2½ or 3 in. (6 to 8 cm.) in size was found on the lateral wall of the cecum and proximal ascending colon. This mass was softer than the usual tumor but considerably harder than a simple inflammatory reaction. On palpation through the normal medial bowel wall, some nodularity and irregularity could be felt throughout this lesion. Careful examination of the terminal ileum revealed only moderate edema of the peritoneal surface but no other intrinsic bowel changes. The transverse colon revealed a small solitary diverticulum but no inflammatory reaction about this. The lesion was palpated carefully, and it was felt that clinically it was impossible to differentiate between a granuloma of the cecum and a carcinoma of this region and that the treatment of choice was right hemicolectomy with ileotransverse colostomy. Accordingly, a right hemicolectomy was performed in the usual manner and a portion of the terminal ileum was excised with the lesion. The distal ileum was then anastomosed in an end-to-end fashion to the remaining open end of the transverse colon. The mesentery was reconstructed for a small distance, and the right peritoneal gutter was obliterated. The abdomen was closed in the usual manner. The patient's postoperative course was relatively uneventful;

oral feedings were begun on the fifth postoperative day and his bowels moved on the same day. The patient was dismissed from the hospital on the 12th postoperative day and returned to work approximately one week later.

Examination on April 17, 1953, revealed that the wound was well healed and the patient's general condition was excellent. He had no further bowel complaints and had no further difficulty of any nature.

*Pathological Findings.**—The specimen consisted of 11 cm. of terminal ileum and 18 cm. of cecum and ascending colon. The appendix was absent. Five centimeters above the cecum on the posterior wall of the colon, slightly toward the medial side, a diverticulum was found (Fig. 2) with an ulcer crater in the base. This diverticulum measured 10 mm. across and 20 mm. in depth. On the outer surface of the posterior portion of the cecum was a dense mass of firm grayish-red tissue with some striated muscle present (Fig. 1). The microscopic sections showed that the mucosal epithelium was ulcerated and the ulcer extended completely through the muscular wall and that there was acute inflammation in the serosal connective tissue (Fig. 3).

SUMMARY

This case is reported in order to emphasize that solitary diverticula of the cecum are not too uncommon and that they may easily be confused with acute appendicitis and carcinoma of the cecum.

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CHYLE CYST OF THE MESENTERY

Brief Review and Report of Case in a Three-Week-Old Infant

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UNUSUAL conditions are worthy of reporting if they are likely to be forgotten in differential diagnosis, with resultant delay or confusion in rendering proper treatment. Such is the case as regards mesenteric cysts which are uncommon enough to be neglected in both teaching and practice.

DEFINITION

Thompson and Chambers [10] provide a reasonable definition of chylangioma as a tumor of lymph vessels which consists of an anastomosing network of lymph spaces supported by thin walls or thick septa. It is lined by epithelium and contains chyle or more inspissated fatty material which is often bloodstained. It may be unilocular or multilocular and, as pointed out by Block,[3] must be differentiated from congenital duplication of the bowel, which it may closely resemble. It may vary in size from a diameter of 3 or 4 cm. to a tumor occupying a large part of the abdominal cavity.

INCIDENCE

Beahrs [1] states that only 9 chylous cysts have occurred in 174 cysts of the omentum, mesentery, and retroperitoneal region in more than 1,000,000 patients at the Mayo Clinic. Slocum [9] reports that 6 cases occurred at the Massachusetts General Hospital from 1900 to 1926; 1 case occurred in over 93,000 admissions at the University of California Hospital, and 1 case in 188,000 admissions to Los Angeles General Hospital. Block [3] found 1 case at Jewish Hospital, Philadelphia, in over 160,000 admissions. Thompson [10] feels that the average of statistics shows the incidence to be about 1 in 100,000 hospital admissions, and Roller [8] summarizes the incidence by stating that mesenteric cysts are the rarest of abdominal neoplasms, and chylous cyst is one of the rarest of mesenteric tumors.

Various authors express different opinions as to the age incidence, Warfield [11] having found, in 129 cases of mesenteric cysts, 9 cases in infants under one year of age and 21 in children from 1 to 10 years, while ages 30 to 40 showed the greatest number, 24 cases. Gross [5] has recently summarized his statistics in 13 cases of chylous cyst and feels it is commoner in childhood than in infancy. He reports five cases under 7 months and eight cases from 2½ to 10 years. Yet Beahrs [2] reports 9 chylous cysts occurring in a total of 174 mesenteric cysts, the patients ranging in age from 24 to 62 years. Most case reports in the literature likewise seem to concern adults.

ETIOLOGY

There is considerable variance of opinion as to etiology of chylous cysts, due probably to the rarity of the cysts and the lack of information about them. Beahrs [2] feels that they are preformed and developmental anomalies, possibly traumatic in a few cases, or occasionally degeneration of lymph nodes. He feels that mechanical obstruction to lymphatic vessels must be discounted, since cysts have not been produced by experimental obstruction of the thoracic duct and because of the extensive anastomosis of lymphatic vessels. The latter view is challenged by Ewing,[4] who feels they are all true chylangiomata due to congenital or acquired obstruction of the lacteals, and by Gross,[5] who postulates their development from misplaced bits of lymphatic tissue which proliferate and then accumulate fluid because they do not possess communication which allow them to drain properly into the remainder of the lymphatic system. Lee [7] advances the theory that cysts form when a chyle-bearing lymph vessel is ruptured and the extravasation is limited by the formation of a wall of granulation tissue. Experimentally, he has not been able to produce cysts by inducing a high degree of obstruction to lymph flow, but has observed, in experimental animals, rupture of a small lymph vessel while material was being injected into the lymph channel, thus supporting the view of cyst development from the extravasation.

Thompson [10] probably best compromises these several viewpoints in feeling that chylous cysts are mostly of embyronic origin though some may be acquired, the former being true chylangiomata and the latter being due to obstruction to lymphatic vessels. A classification which embraces the various etiological theories is proposed by Beahrs,[2] as follows:

A. Embryonic or developmental

B. Traumatic or acquired

C. Neoplastic

D. Infective and degenerative

SYMPTOMS, SIGNS, AND DIAGNOSIS

There are no pathognomonic symptoms or signs of chylous cysts other than the general ones which may attend the presence of an abdominal tumor. Thus, painless tumor may be the only complaint. Depending on the size, location, and pressure upon other viscera, there may be partial intestinal obstruction, peritonitis secondary to cyst rupture, or incarceration of the cyst in the pelvis. Symptoms most commonly observed are crampy pain, malaise, anorexia, nausea and vomiting, and fatigue and weight loss. The cyst may appear as a slowly enlarging affair; it may cause recurrent attacks of mild to moderate abdominal pain and associated symptoms, or it may precipitate an acute "surgical abdomen."

The picture may be confused with that of duplication of the bowel, ovarian cyst, retroperitoneal tumor or cyst, pancreatic cyst, hydronephrosis, bowel tumor, kidney tumor, or intussusception, and in this regard, as emphasized by Hinkel,[6] x-rays may be of value in that the tumor may be palpated under the fluoroscopic screen after barium has been administered by mouth or rectum. In the flat plate the cyst usually appears as a gasless mass displacing abdominal contents.

The diagnosis is not usually made preoperatively but should always be considered by the surgeon when faced with an abdominal condition not easily explained by ordinary pathology. As Roller [8] states, the mortality is higher than it should be and the diagnosis is uncertain before operation: Both should be improved.

TREATMENT AND RESULTS

It should go without saying that preoperative deficiencies should be corrected as far as possible. It is wise to insert a Levin or Miller-Abbott tube in anticipation of possible ileus or the necessity of resection. As in the case of most cysts, enucleation or excision should be given preference if at all possible. This may be a relatively easy task or a most tedious one and may involve intestinal resection. Great care must be given to preservation of the blood supply to the bowel, particularly in the infant in whom the vessels are thread-like and delicate. The viability of the bowel must be frequently observed during the course of the dissection. Hemostasis must be exact to prevent hematoma formation between the leaves of the mesentery. It may be impossible to remove completely all bits of tissue close to the root of the mesentery, but apparently a small remnant may be left without fear of recurrence. Some authors have recommended phenolization of this tissue, but this would seem to be not without danger, particularly in infants. No cases of recurrences have been noted in the literature, however treated. Marsupialization or simple drainage are not recommended as worthy procedures, though a few successful cases have been reported.

Mortality rates have varied from 22 to 50%, though Gross [5] reports only 2 deaths in 12 cases with operation, and these had associated volvulus. With better recognition of this entity and with modern emphasis on preoperative and postoperative care, as well as gentle handling of tissues, attention to hemostasis, and the use of indwelling tubes, it may well be that the mortality in recent years has been considerably improved.

REPORT OF A CASE

P. P., aged 20 days, a white infant girl, was admitted to Princeton Hospital at 7:30 p. m. on July 5, 1953, with a chief complaint of blood in the stool and crying. Delivery had been uncomplicated at term, with formula feeding without problems. The baby was well until 24 hours prior to admission, when she apparently developed severe abdominal colic, with straining to pass a normal yellow stool. She was fussy thereafter, crying every hour or so. She was reluctant to take her formula, and 11 hours prior to admission passed mucous and blood by rectum. This was followed by four or five bloody stools. There was no vomiting. She was then seen by her pediatrician, who advised hospitalization.

Initial examination showed a well-nourished infant in obvious distress, crying frequently. The skin was moist with good elasticity. There was a readily palpable mass, visible at times, with visible peristaltic waves, in the left abdomen. Faint borborygmi were audible. The right lower quadrant felt somewhat empty. Rectal examination was negative, but there was a considerable amount of blood on the examining finger. The pulse rate was 160, the temperature normal. Initial blood count showed 5,550,000 RBC with 15.5 gm. of hemoglobin per 100 cc. White blood cell count was 14,500 with 5 juveniles, 21 stab forms, 35 segmented cells, 34 lymphocytes, 3 monocytes, and 3 eosinophiles.

Scout film of the abdomen showed a step-ladder appearance of small intestinal loops in the right lower quadrant, with some gas in the transverse colon, sigmoid, and rectum. There was a mass in the left upper quadrant, irregular in outline. The roentgen appearance suggested a mechanical ileus.

The initial impression was possible volvulus or intussusception, though the x-ray gas pattern did not suggest the latter. It was difficult to account for the mucous and bright blood by rectum. The baby was given 100 cc. of dextrose in isotonic sodium chloride solution intravenously and prepared for laparotomy.

The infant was anesthetized with vinyl ether (Vinethene), open-drop ether, and oxygen, and a left transverse incision was made ½ in. (1.3 cm.) above the umbilicus. As the peritoneum was opened, a large amount of milky fluid immediately exuded. This was aspirated and cultured. A large mass was then visualized and palpated, cystic in nature, bluish in color, occupying nearly the entire left side of the abdomen. The incision was then enlarged to the right and the intestinal loops packed off. Further investigation revealed the tumor to lie within the leaves of the mesentery of the distal ileum, which was adherent as it coursed over the top of the cyst. The bowel was flattened at this level but was not particularly distended above nor deflated below. There was a perforation near the root of the mesentery from which the fluid was leaking. By gentle sharp and blunt dissection it was possible to excise the cyst except for a small remnant of tissue near the base of the mesentery, where it was felt that further dissection might injure the blood supply. The tiny blood vessels were tediously dissected free from the cyst, with constant observation of the color of the bowel. After the cyst was removed, the bowel seemed viable, and hemostasis was good. It was not felt that the vessels had been sufficiently abused to cause thrombosis; consequently, resection was not performed. The abdomen was closed in layers with fine nonabsorbable surgical (silk) sutures and the baby returned to bed in good condition.

Postoperatively, alternate clyses of 100 cc. of dextrose in saline and dextrose in water were administered every four to six hours. The following day there was slight vomiting, but the baby passed a considerable amount of gas. The abdomen was soft, the color good, the temperature 101 F., and the white blood cell count had fallen to 7,100, with a normal differential distribution. Late in the day there was more vomiting; consequently, a tube was placed in the stomach for suction. The second day the baby had a liquid bowel movement, normal in color, and on the third day there were several good bowel movements, and the tube was removed. Oral feedings of dextrose water were begun, and the feedings rapidly increased to skimmed milk and house formula. The temperature was normal by the fourth day, and convalescence was then rapid. The sutures were removed on the seventh day and the infant discharged on the ninth postoperative day.

Unfortunately, the collected fluid and cyst were misplaced enroute to the laboratory; consequently, no microscopic report was available. However, there seemed to be little doubt as to the diagnosis.

The baby was seen in follow-up at 6 months of age, at which time the incision was well healed and the child appeared normal in every respect. There was no evidence of recurrence.

COMMENT

This case is unusual not only because of the age of the patient but because of the character of the symptoms. Bloody stools have not been mentioned as occurring in this entity, yet this was the predominate sign, along with the mass and pain. It is surmised that extrinsic pressure upon the bowel wall, with resultant venous congestion and oozing from the mucous membrane, was the cause of the bleeding, since no inherent pathology within the bowel lumen was noted at operation.

Preoperative diagnosis was not made, as is the usual situation, but operation was fortunately undertaken with a successful result. The case is reported to emphasize the existence and nature of this condition so that it may come to mind oftener in differential diagnosis of obscure abdominal tumors.

SUMMARY

Mesenteric cysts are among the rarest of abdominal tumors and chylous cysts are probably the rarest of these.

The etiology of these tumors is somewhat confused owing to the paucity of cases and the inability to reproduce them experimentally.

The diagnosis rests on detection of an abdominal mass, with its resultant symptoms, coupled with the clinician's awareness that it may be a mesenteric cyst.

The treatment is surgical, preferably excision of the cyst with resection if necessary.

A case occurring in a 20-day-old infant is reported, with successful excision of the cyst and no recurrence at six months.

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ROENTGEN CHANGES IN TRANSIENT SYNOVITIS OF THE HIP JOINT

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THE CONDITION referred to as transient synovitis of the hip joint is not infrequently encountered in clinical practice. We, in the past year have seen six cases in our clinical and hospital material. The medical literature, however, has very few reports of this entity, and the radiological literature contained none until a recent article by Drey presented some of the roentgen findings in a case which he had the opportunity of following. We are presenting two illustrative cases which demonstrate the salient features of this disease.

Belmonte, Bradford and Lovett, Butler, Finder, and Miller have contributed papers on the subject. Lucas, in 1948, and Edwards, in 1952, reviewed the entity of transient synovitis of the hip joint and their valuable contributions summarized the present knowledge of the subject. Other names by which this condition has been identified are transitory coxitis, coxitis serosa seu simplex, coxitis fugitive, and transitory arthritis of the hip.

The condition affects children usually below the age of 10 years, with boys being affected more frequently than girls. The presenting symptom is a limp produced by pain in the hip. The hip is held in adduction and flexion which decreases intra-articular tension. All ranges of movement of the hip are limited, and all the muscle groups about the hip disclose spasm. Extension of the leg is severely resisted because of marked accentuation of pain.

Usually there is a low-grade temperature elevation. Laboratory data is otherwise not of help except in a negative manner. Thus a negative serology and a negative reaction to tuberculin are important in excluding syphilis and tuberculosis, which must be differentiated from transient synovitis.

Drey points out that swelling of the synovial membrane of the hip is associated with swelling of the musculature surrounding the hip joint. Among the muscles thus affected are the obturator internus, the gluteus minimus, and the iliopsoas. The muscular swelling may be recognized as a thickening of the muscle shadow and by erasure of the shadow of the intermuscular septum which is usually seen as a radiolucent line. Comparison of the affected side with the normal side helps in recognition of these criteria.

In addition to the muscle swelling we have noted other roentgen findings which help in the diagnosis. If good detail films are obtained, one can easily discern the swollen joint capsule of the affected hip as it bulges laterally.

If the process in the hip is followed by serial x-ray films, the muscle swelling and the distention of the joint capsule will subside quickly, just as the clinical features quickly disappear.

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At no time is there any recognizable deossification of the bone structure. This finding helps in excluding many serious diseases of the hip.

We have found it rather easy to identify the distended joint capsule and the thickened obturator internus. The swelling of the iliopsoas muscle and the gluteus minimus has not always been recognizable in our experience. Perhaps now that Drey has called attention to these changes a better knowledge of what to look for will reveal these findings more frequently.

Edwards aspirated fluid from the hips of two of his patients, but aside from slight xanthochromia no unusual changes were noted in the fluid constituents. A study of pathological tissue was never described because of the benign course of the disease and consequent difficulty in getting operative or postmortem material.

Previous to Drey's article, authors have considered roentgenography as offering little or no information in the diagnosis of transient synovitis. There are, however, important changes in the radiographs which can be of great aid in the diagnosis. The roentgen changes are illustrated in the following two cases.

CASE 1.—A. B., a 5-year-old boy, was seen on Dec. 18, 1951, by his pediatrician, with pain in the left hip and the lateral aspect of the left thigh and left knee. The pediatrician noted restriction of motion in all planes and directions, and he made a provisional diagnosis of Legg-Calvé-Perthes disease.

The patient was referred to an orthopedic surgeon who noted a slight elevation in temperature, 100.6 F. and a normal pulse rate. The patient had a definite limp, and physical examination again confirmed the restriction of movement of the hip in all directions. X-rays of the hip (Fig. 1) disclosed bulging of the joint capsule and thickening of the obturator internus shadow. There was no visible deossification of the bone structure. The iliopsoas shadow near its insertion in the femur was perhaps slightly swollen. The gluteus minimus could not be clearly made out. Transient synovitis of the hip was offered as a likely diagnosis with a suggestion to recheck the hip after a short interval to exclude more serious disease.

The patient was put to bed and told to return in one week. The pain subsided markedly by the third day and was completely gone by the fifth. On his return visit there was no evidence of limp. A full range of movement was restored in the hip; muscle spasm was gone. A recheck x-ray study disclosed complete restitution to normal.

The etiology in this case remains obscure, since there was no indication of trauma or of infection elsewhere in the body.

CASE 2.—N. L. E., a 6-year-old white girl, was seen by her family physician because of pain in the left hip of five days' duration and a limp of two days' duration. There was no history of trauma or infection. Temperature, pulse rate, and respiration were normal. Physical examination revealed limited motion in all planes of the left hip joint. She was referred for x-ray studies of the hip (Fig. 2). The roentgenograms revealed swelling of the joint capsule and of the obturator internus. No distinct changes could be made out in the gluteus minimus or in the iliopsoas. There was no deossification of the bone structure and no other visible abnormality. Transient synovitis of the hip was suggested as a strong possibility, and clinical supervision and a follow-up x-ray examination were advised to exclude a more serious entity. The patient was restricted to limited activity, and the symptoms and signs disappeared within three days. X-ray examination two weeks later disclosed a perfectly normal hip joint.

The condition runs a benign course, subsiding as a rule within three days, although in one of Edwards' cases it lasted as long as three weeks. Healing is complete with no sequelae and in no instance has a recurrence been reported.

Transient synovitis is important to the physician because it must be differentiated from several other serious and disabling maladies of the hips. Osteomyelitis adjacent to the hip joint may be confusing particularly early in the disease. How-

Fig. 1 (Case 1).—*A*, left hip at the height of the clinical picture. Arrows point to bulging joint capsule (lined in pencil), and to thickened obturator internus muscle. The iliopsoas muscle shadow near this insertion appears more prominent on the diseased side. *B*, same hip one week later shows complete resolution with a normal configuration of the joint capsule and obturator internus. There is no deossification of the bones in either film. The iliopsoas muscle shadow seems less prominent than in *A*.

Fig. 2.—*A*, left hip shows the same findings as in Case 1. Bulging of the joint capsule, thickening of the shadow of the obturator internus, and a normal degree of ossification of the bone structure. The iliopsoas muscle is not distinctly abnormal. *B*, same hip two weeks later shows complete restoration to normal.

ever, persistence of symptoms, the elevated leucocyte count and the hyperemic deossification of the bone, plus destructive changes in the bone at a later date, are sufficient to clarify the diagnosis.

Tuberculosis can be differentiated by history of exposure to tuberculosis, tuberculin test, and x-ray findings of deossification of the bone, joint-space narrowing, and destructive changes in the bones. Guinea pig inoculation of aspirated joint fluid may be necessary in some cases to establish the diagnosis of tuberculosis.

Syphilis can be excluded by searching for other stigmata of syphilis, and by checking the serology. The x-ray changes of syphilis, namely, the osteochondritis, metaphysitis, and periostitis, also are quite helpful.

Legg-Calvé-Perthes disease is best differentiated by the characteristic x-ray changes in the capital epiphysis. The chronic course of Legg-Calvé-Perthes disease and absence of fever are diagnostic.

In slipped epiphysis the x-ray findings are conclusive in establishing the diagnosis. Other conditions such as rheumatic fever and rheumatoid arthritis usually have other joints involved and have other clinical stigmata which offer a ready diagnosis.

The etiology of transient synovitis is unknown, but it has given rise to considerable speculation. Butler thought that any one of several factors, including trauma and inflammation such as measles, scarlet fever, tonsillitis, and influenza, may be involved in the causation.

Edwards feels that in addition to trauma and infection, allergy may play an important causative role. He postulates an allergic hypersensitivity of the synovial membrane to an infection elsewhere in the body.

The disease is self-limited and of short duration and usually clears up within three days. Rest of the involved lower extremity frequently is sufficient treatment. In severer cases traction may be necessary.

SUMMARY

Transient synovitis of the hip is not infrequent in children. It follows a benign self-limited course, but it must be differentiated from serious hip disease. X-ray study of the involved hip reveals no deossification of the bones of the hip, swelling of the joint capsule, swelling of the internal obturator muscle, swelling of the iliopsoas muscle and the gluteus minimus in some cases, and rapid subsidence of the soft tissue changes.

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RETROPHARYNGEAL SOFT TISSUE SWELLING DUE TO WHIPLASH INJURY

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THE SIGNIFICANCE of swelling of the retropharyngeal soft tissues in the presence of fracture and/or dislocation of the cervical spine has been stressed by Heublein.[1] But to our knowledge, swelling of the retropharyngeal soft tissues due to whiplash injury without radiographic evidence of fracture and/or dislocation has not previously been reported.

The "whiplash" injury is usually incurred from automobile accidents and is due to sudden deceleration. Nine cases of marked swelling of the retropharyngeal soft tissues due to whiplash injury comprise the major basis of this report.

Current analyses of roentgenograms of the cervical spine, whether by radiologists, neurosurgeons, or orthopedists, disregard all forms of swelling of the retropharyngeal and retrolaryngeal soft tissues. Their attention apparently has been focused on search for fractures and/or dislocation, some authorities attaching various degrees of significance to the cervical curve.

A brief review of the normal limits of soft tissue spaces of the neck is pertinent to this discussion. In "Diagnostic Roentgenology" (Vol. 2) by Ross Golden, Barton Young states:

> It is extremely important that the roentgenologist be aware of the normal width of the soft tissue zones—because of the prevalence of phlegmon and tumors in these regions. For a number of years we have made use of measurements set forth by Hay in his splendid monograph on the neck to determine the approximate normal size of these soft tissue spaces.

The measurements of Hay are presented in Table 1.

Since the advent of antibiotics, the prevalence of "phlegmon" in these areas has been practically negligible. However if "trauma" is added to the list, the significance of the use of this Table becomes apparent.

With use of the measurements of Hay, the roentgenograms of 121 cervical spines taken at Kern General Hospital were reviewed. (Two cases of fracture of the neural arch not associated with widening of the retropharyngeal soft tissue space were discovered.) In addition to our 9 instances of swelling of the retropharyngeal soft tissues due to whiplash injury, we found 10 others due to different trauma: (*a*) with fracture (Case 1), (*b*) occurring after reduction of fracture and/or dislocation but not present at time of injury, (*c*) other traumatic causes (gunshot wound, injury to chest, fracture to mandible).

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Table 2 presents in tabular form the relative thickness of the retropharyngeal soft tissue space (according to the measurements of Hay) in various types of injuries.

The pathogenesis of retropharyngeal swelling has been subdivided into (*a*) edema and (*b*) hemorrhage. We have postulated that when the swelling disappears within 72 hours, it was caused by edema. A swelling that persisted beyond 72 hours was classified as due as hemorrhage or a combination of edema and hemorrhage.

Representative cases of our study are presented.

Case 1.—S. J. (automobile accident). On admission the patient complained of considerable pain in the cervical area. Roentgenograms revealed fracture of the anterior margin of the second cervical vertebra associated with marked swelling of the retropharyngeal soft tissues;

Table 1.—*Upper Normal Limits of Soft Tissue Spaces of Neck* *

Age, Yr.	Postpharyngeal Soft Tissue, C†		Postpharyngeal Soft Tissue, C†		
0-1	1.5		2.0		
1-2	0.5		1.5		
2-3	0.5		1.2		Postventricular
3-6	0.4		1.2		
6-14	0.3		1.2		
Adult	Male 0.3 C	Female 0.3 C	Male 0.7 C	Female 0.6 C	Postericoid

* This table is reproduced with modifications by permission of Williams & Wilkins Company, publishers of "Diagnostic Roentgenology," by Ross Golden.
† C is anteroposterior dimension of fourth cervical vertebral body measured at its middle.

Table 2.—*Degree of Retropharyngeal Soft Tissue Swelling in Various Types of Injuries* *

Percent of Retropharyngeal Soft Tissue Swelling	Types of Injury			
	Whiplash	Fracture	Occurring Post-Reduction	Other Causes (Gunshot, Etc.)
0-50	8	2	..	..
50-100	..	3	2	2
Over 100	1	1	..	..

* In two cases of fracture of neural arch there was no swelling.

the soft tissue space measured 11 mm. as compared with 17 mm. for the thickness of the fourth cervical body (Fig. 1*A*). The swelling receded rapidly and was within normal limits four days following admission (Fig. 1*B*).

Case 2.—R. H. (automobile accident). This patient entered the hospital complaining of pain in the cervical area. Roentgenograms at the time of admission failed to reveal evidence of fracture. There was loss of the normal cervical lordotic curve, and swelling of the retropharyngeal soft tissues was present. The patient was dismissed from the hospital without follow-up examination until called back to aid in the completion of this study.

The follow-up film, taken 10 months later, revealed entirely normal thickness of the retropharyngeal soft tissues and no radiographic evidence of abnormality of the cervical spine.

Case 3.—R. W. (automobile accident). Roentgenograms at admission failed to reveal evidence of fracture. The soft tissues of the retropharyngeal space measured 14 mm. as compared with 19 mm., the width of the fourth cervical vertebra. Follow-up films obtained seven months after the original injury disclosed the soft tissue space had returned to normal.

Case 4.—C. C. (automobile accident). On admission the patient complained of considerable pain in the cervical area. Roentgenograms at that time did not reveal any evidence of fracture,

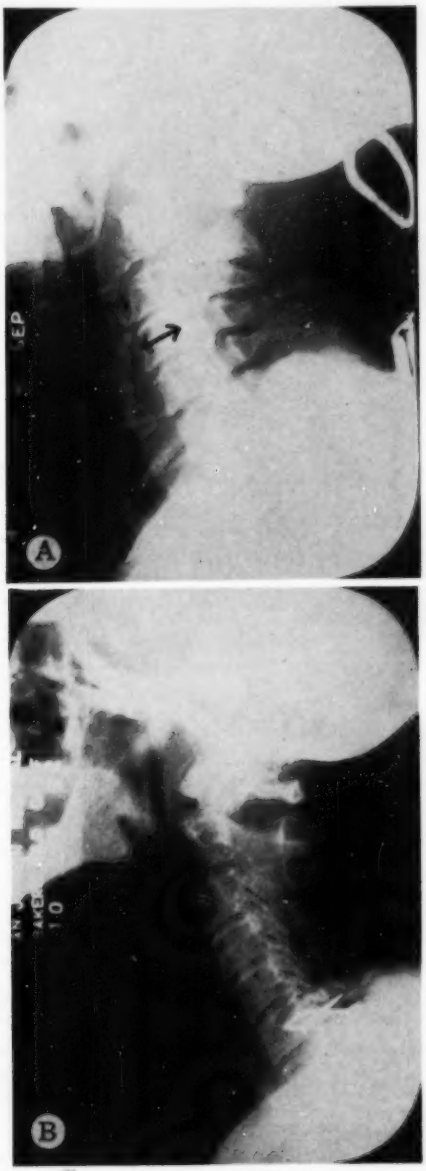

Fig. 1 (Case 1).—*A*, roentgenogram taken immediately after injury, showing maximum soft tissue swelling. *B*, roentgenogram taken four days later. Swelling is gone and soft tissue space is normal.

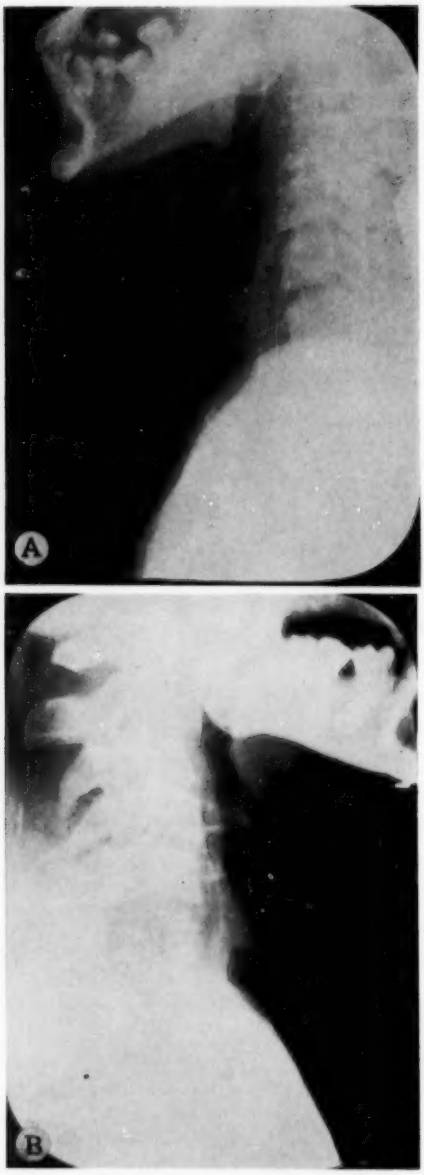

Fig. 2 (Case 4).—*A*, roentgenogram taken immediately after injury. Note increase in thickness of retropharyngeal soft tissue space. *B*, roentgenogram taken 10 days after injury. Soft tissue space returned to normal width.

but the retropharyngeal soft tissue thickness was 9 mm. as contrasted with 12 mm., the width of the fourth cervical vertebra (Fig. 2*A*). The patient was placed in a felt collar; she made good recovery without sequellae. Serial films were taken; the swelling slowly but gradually subsided until 10 days after the accident it reached a minimum of 6 mm. (Fig. 2*B*).

COMMENT

Our observations indicate that the thickness of the retropharyngeal soft tissues may be of value in further determining the exact degree of trauma both to the soft tissues and to the bony component of the cervical vertebrae. In one instance of marked swelling of the retropharyngeal soft tissues in the absence of fracture on routine views, laminograph studies revealed a fracture of the dens.

Davis [2] regards straightness of the cervical spine in the absence of fracture or dislocation to be an indication of hidden cervical spine damage. We feel that swelling of the retropharyngeal soft tissues is also significant, particularly in the absence of radiographic evidence of fracture, and is indicative of hemorrhage and/or edema in the longitudinal ligaments. This is of primary importance in the whiplash injury type of case, and further studies may indicate that patients with swelling of the retropharyngeal soft tissues warrant a longer period of immobilization.

We have not had an opportunity to follow these cases over a period of years, but the radiographic findings suggest that this type of injury might (*a*) possibly be related to late degenerative disk disease of the cervical spine or (*b*) be a causitive factor in the formation of osteophytes which are so characteristic of late results of trauma.

Our findings suggest that such injuries might be of significance years later following the injury; therefore, we feel that further study is warranted.

CONCLUSIONS

After any severe injury to the neck, routine views of the cervical spine should be secured as soon as possible; if there is any indication of swelling of the retropharyngeal soft tissues, a repeat lateral view in 24 hours is indicated.

If there is swelling of the retropharyngeal soft tissues in the absence of fracture, multiple views (laminographs) may be indicated.

If the swelling is transient, we have assumed the process is one of edema; if the process is slowly resolving, it is thought to be due to hemorrhage or to a combination of edema and hemorrhage.

The absence of swelling of the retropharyngeal soft tissues may be an aid in determining whether an injury to the cervical spine is recent or old.

Since swelling of the retropharyngeal soft tissues occurs in the absence of fracture, and in our series was associated with approximately 25% of all whiplash injuries, it may be the only concrete evidence of injury. It would therefore be an index to the degree of trauma and of major importance from the medical-legal aspect.

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TRENDS IN ORTHOPEDIC SURGERY

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THE UNSOLVED FRACTURE

IN 1934 Kellogg Speed used the term "the unsolved fracture" in a discussion of the intracapsular fracture of the neck of the femur. Since that time so many changes have been made that Dr. H. R. McCarroll [1] has considered this to be a suitable time to review his experiences and to restate the problem as it exists today.

Internal fixation is the method that is almost universally accepted as the preferred procedure in the early treatment. In McCarroll's series of 203 cases, aseptic necrosis occurred in 60 and nonunion in 50; hence, the results leave much to be desired despite the fact that the fracture may be seen early, accurately reduced, and adequately pinned.

Some type of reconstructive procedure is called for if a complication such as aseptic necrosis, displacement of fragments, or nonunion occurs. In this study the results obtained by employing reconstructive methods were appraised. Various types of subtrochanteric osteotomies change the angle of the fracture line from the vertical to more nearly horizontal and thus overcome nonunion, but they will not control aseptic necrosis. Twelve cases treated by osteotomy failed to produce a satisfactory end-result. In nonunion cases the absorption of the femoral neck makes the anatomic structures unsatisfactory for the use of Smith-Petersen cup arthroplasty. The Colonna arthroplasty, which places the trochanter within the acetabulum and reattaches the abductor muscles to the femoral shaft at a lower level, produced a fair outcome in five cases. Bone grafts placed across the site of the fracture were not successful in the five cases attempted. Moore's cartilaginous cup arthroplasty did not serve the purpose in six cases in which it was done. This study concludes that the treatment of the complications encountered in fractures of the neck of the femur has remained unsatisfactory despite the employment of all the recommended reconstructive procedures. There may be some hope in the use of the replacement type of prosthesis, but unfortunately no one person has had sufficient experience to state with certainty that its use will turn out well in the majority of cases.

Because the study of these many reconstructive procedures for the hip have occupied the attention of orthopedic surgeons during the past few years, one must deduce that most surgeons feel that the present treatment of this fracture is ideal and no further study is needed.

This attitude is a defeatist attitude, and Dr. James Dickson,[2] in a presidential address at the annual meeting of the American Orthopaedic Association, held in

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Hot Springs, Va., on June 27, 1953, protested vigorously against it. He is of the opinion that too many surgeons in the face of impending nonunion or aseptic necrosis have failed to make a renewed effort to secure union but have turned instead to the salvage procedure of using a replacement prosthesis.

It is his conviction that revascularization of the head fragment will take place and much better results will be obtained if the following principles are followed:

1. Anatomical reduction.

2. Absolute and complete immobilization. In this respect a Jewett type trifin nail placed low in the neck so that it rests on the calcar femorale with its added shaft fixation providing increased stability is considered to be superior to the Smith-Petersen nail which does not have the angle support.

3. Close supervision of patients and repeated x-ray studies during the first year in order to uncover the earliest change in position or absorption of bone. If such occurs, surgical removal of scar tissue at the fracture site and the insertion of a cancellous bone graft will prevent the avascular change if the procedure is done before the process has become irreversible.

4. Subtrochanteric osteotomy to eliminate the shearing stress combined with the cancellous bone graft across the fracture site in the more advanced cases of nonunion in which the fracture is not impacting and the head assumes a varus position.

CENTRAL AGENCY TO COORDINATE ORTHOPEDIC RESEARCH

The General Motors Company has its Technical Center where all problems of research are planned, with the utilization of expert advice on each phase of the problem. Realizing that this modern approach could be adopted with benefit by the members of the medical profession in order to collect, compare, evaluate data, and integrate individual efforts, Dr. Dickson has called for the establishment of such an agency as a joint enterprise of the Association and of the Academy of Orthopaedic Surgery.

NATIONAL SERVICE LABORATORIES, INC.

Dr. Joseph S. Barr [3] has centered our interest on a variable factor which can determine the success or failure of a surgical procedure. This factor is related to the physical and biological properties of the surgical material left in the body. These materials range from absorbable surgical (gut) sutures to nails, plates, Vitallium cups, acrylic prosthetic replacements, and oxidized cellulose. Very little information is available on the incidence of operative failure due to defective surgical material, although it is common knowledge that failures occur due to weakness of the material, foreign body reaction, and biological incompatability.

At present the manufacturers of surgical materials furnish little specification data; consequently, it remains for the human guinea-pig technique method to test the claims of the manufacturer. This unhappy situation is inexcusable in view of the availability of scientific testing methods.

Dr. Barr recommends that all surgical materials used within the human body be tested so that their physical and biological properties are known in order to guide the surgeon in his choice of materials. The design of surgical devices, likewise, should be analyzed mechanically and biologically to ensure that they will fulfill their function in the human body.

In order to do this, it is his suggestion that the medical profession unite with the manufacturers and distributors of surgical equipment to form a nonprofit organization with the authority to test surgical materials, to conduct and encourage research in surgical engineering, to hold patents and utilize income derived for further investigation, and to standardize sizes and types of devices to assist the manufacturers to produce better surgical materials at decreased cost.

Representatives of the surgical manufacturers, the American Academy of Orthopaedic Surgeons, the American Medical Association, and the American College of Surgeons have expressed a willingness to work on this problem. It is hoped that "National Surgical Laboratories, Inc." may be the outcome of their deliberations.

IPRONIAZID IN THE TREATMENT OF BONE AND JOINT TUBERCULOSIS

The use of iproniazid (1-isonicotinyl-2-isopropylhydrazine) in the treatment of bone and joint tuberculosis by Dr. Bosworth [4] and his associates indicates that it has a more rapid and specific effect where bone and joint lesions are concerned than does isoniazid. In the cases studied there were 98 lesions, of which 45 healed, 37 were improved, and 16 unimproved. Although five patients died while under treatment, none of them showed evidence of iproniazid toxicity. The general benefits observed were gain in weight, closure of sinuses, decrease in the size of closed abscesses, and a progressive healing of open lesions.

While under treatment, patients showed many evidences of toxicity, such as dizziness, tremor, positive Babinski, hypertonic reflexes, clonus, euphoria, drowsiness, headaches, tinnitus, and phychosis. All of these symptoms are mild except psychosis; however, this appears rarely and can be controlled by dosage reduction.

The average dose used is 4 mg. per kilogram of body weight. If severe hypertonicity or clonus develops, the dose is reduced to 2 mg. per kilogram of body weight, and then it is gradually increased to between 3 to 4 mg. per kilogram. The concentration of the drug in the serum must be kept to a certain minimum level to secure desirable results. If combined with good medical care, dietary care, and surgical treatment, Dr. Bosworth feels that iproniazid will prove to be the drug of choice in the treatment of tuberculous bone and joint lesions.

THE JUVENILE AMPUTEE

The juvenile amputee is singularly free from the problems which beset the adult. This was revealed in an analysis of 192 cases of amputations in children made by Drs. G. T. Aitken and C. H. Frantz [5] at the Juvenile Training Center, in Grand Rapids, Mich.

Scar formation was not a problem, and revision of the stumps was never necessary because of poor placement of the scar. No complications occurred as a result of spur formation, although spur formation was a frequent finding. Neuromata produced symptoms sufficient to justify surgical treatment in only five cases. Children appear to adapt themselves well to short stumps, and these can be satifactorily fitted. Overgrowth of bone is the phenomenon peculiar to the younger amputee. The humerus is more likely to overgrow than are the bones of the forearm. The femur rarely overgrows enough to require surgical revision. The fibula tends to overgrow the tibia. This complication should be anticipated at the initial amputation by sectioning the fibula higher than the tibial stump or by effecting a synos-

teosis operation. Phantom limb does not occur in children whose amputations are done under the age of 10. In the older child, although the phantom-limb impression may be present, there is no associated pain.

In the congenital amputation group where there is a hypoplastic anomaly below the knee the modified conventional appliances, such as molded leather foot buckets, will disguise abnormalities, but they are much less satisfactory than the conventional appliances used after disarticulation. In this instance the disarticulation is the recommended procedure because the thighs remain equal in length.

The optimum time for applying a prosthesis to a child with an amputation of the upper extremity appears to be at age 4 years, one year prior to formal schooling. Younger children can not use it, but at age 4 years the mental development and kinesthetic sense are sufficient to allow the patient to put the appliance to proper use.

INTRAMEDULLARY NAILING OF FRACTURES OF THE TIBIA

The use of intramedullary nails for fractures of the tibia has not been greeted with much enthusiasm because the snags are many and the technical details not firmly established. The anatomical structure of the medullary cavity of the tibia provides one of the difficulties. The medullary canal is large and wide at the upper and lower end, while the central portion is small and narrow ; consequently, in order to provide stability the nail must be the exact length, size, and curve to give three-point support against the opposing cortices of the walls of the tibial shaft.

Because of such technical difficulties, most orthopedic surgeons have reserved medullary fixation of the tibia for specific indications, such as proposed by Dr. J. E. M. Thomson,[6] of Lincoln, Neb.:

1. Transverse fractures of the middle third of the shaft which do not yield to conservative methods of reduction.

2. Severely comminuted simple fractures in which by use of the nail full length and alignment can be maintained.

3. Certain compound fractures of the tibia particularly in the middle third and those in which there has been loss of soft tissue. After débridement these fractures can be held in good position by the nail while plastic procedure on the soft tissues and skin can be carried out.

4. Old neglected fractures of the midshaft with nonunion or malunion. In these the fibrous tissue between the fragments must be excised, the medullary canal reamed out, end-to-end contact secured, and after the medullary nailing is performed, cancellous bone chips are packed about the fracture site and an osteotomy of the fibula is done.

The practical details of medullary nailing of the tibia are gradually being understood and the obstacles overcome. As this occurs, the trend in many clinics is to employ the procedure as the form of definitive treatment of fresh fractures in the area between the midshaft and the junction of the middle and distal thirds of the tibia.

This attitude is reflected in a recent comparative evaluation of the treatment of fresh fractures of the tibia by closed reduction, plate fixation, and medullary nailing made by Drs. J. O. Lottes, J. L. Hill, and J. A. Key,[7] of St. Louis.

In this review the treatment by intramedullary nailing was much superior to both the closed-reduction method and the open reduction with plate-fixation technique in respect to time spent in the hospital, in time elapsed before full weight-bearing without support, and in frequency of complications. This study suggests that, in experienced hands, this method of treatment improves the end-results in fractures of the midshaft of the tibia.

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Scientific Exhibits

BODY TEMPERATURE DURING ANESTHESIA

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HEAT RETENTION (also called heat stroke, or heat pyrexia) arises from a disturbance of the normal balance between heat gain to the body and heat loss. The principal factors concerned with heat balance are shown in Figure 1.

Of the four physical processes by which heat loss is mediated, evaporation is the most important in hot environments. When air temperature becomes equal to skin temperature (about 94 F. dry-bulb temperature), radiation and convection

Average Air Temperature Under the Surgical Drape

Room Temperature, F.	Temperature Under Drape, F.	Patients, No.	Measurements
75-79.5	89	22	586
80-84.5	93	30	1,194
85-89.5	96	11	419
90-95	98	13	309

are no longer avenues of heat loss, and cooling is effected solely by evaporation. It can be seen from the Table that above a room temperature of 85 F., the temperature in the patient's immediate environment, i. e., under the drapes, is 96 F. or more. Hence, above this room temperature it is clear that in any patient in whom the sweating mechanism fails heat retention will almost certainly develop.

The signs of heat retention occurring in the surgical patient are shown in Figure 2. A progressive rapid rise in body temperature is characteristic. Frequent measurement of the patients' temperature should be made during hot weather. The posterior nasal temperature is convenient to measure and is satisfactory. In addition, cessation of sweating should be regarded as an urgent danger signal.

Also shown are recommended preventive measures. Chief among these is to postpone prolonged elective operations during very hot weather. As will be shown (Fig. 4), the incidence of heat retention is directly related to the duration of surgery as well as to the ability to tolerate the environment as measured by the wet-bulb temperature. In patients requiring emergency surgery during hot weather, the lightest possible draping should be used. Asepsis is actually maintained more satisfactorily because evaporation is facilitated and the patient is less likely to sweat through and contaminate the drape. Special care is necessary for children, as the factors of small body mass, high internal heat production, and immature thermoregulatory responses make heat retention a particular hazard.

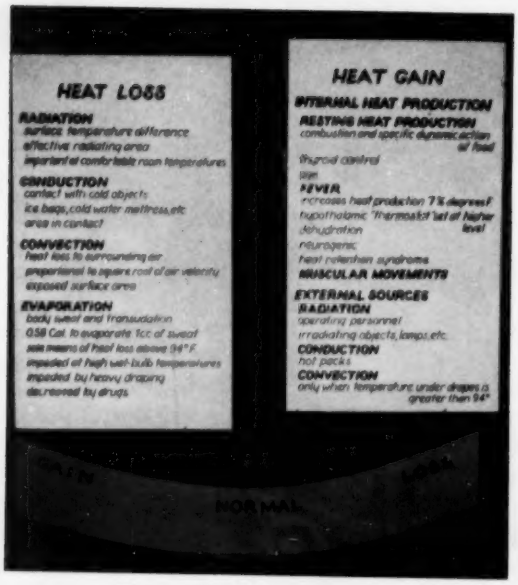

Fig. 1.—Heat-balance equation.

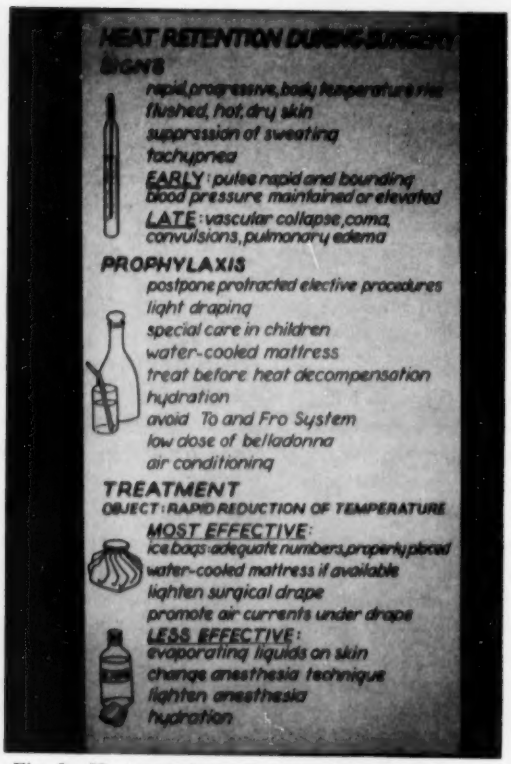

Fig. 2.—Heat retention: signs, prophylaxis, treatment.

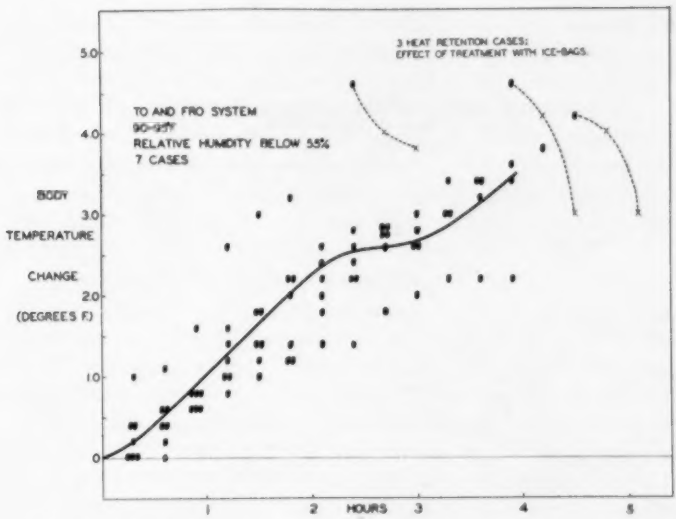

Fig. 3.—Chart showing effect of ice-bag therapy in three heat-retention cases.

AVERAGE FAHRENHEIT TEMPERATURE OBSERVED IN VARIOUS LOCATIONS IN THREE DIFFERENT ANESTHESIA SYSTEMS.

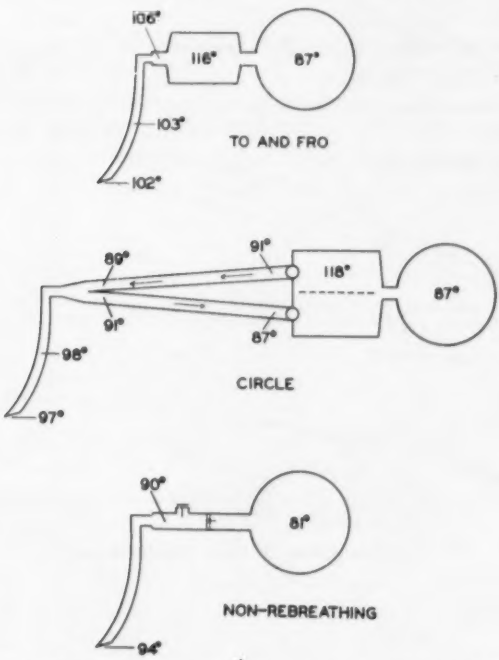

Fig. 4.—Body-temperature rise at various wet-bulb conditions.

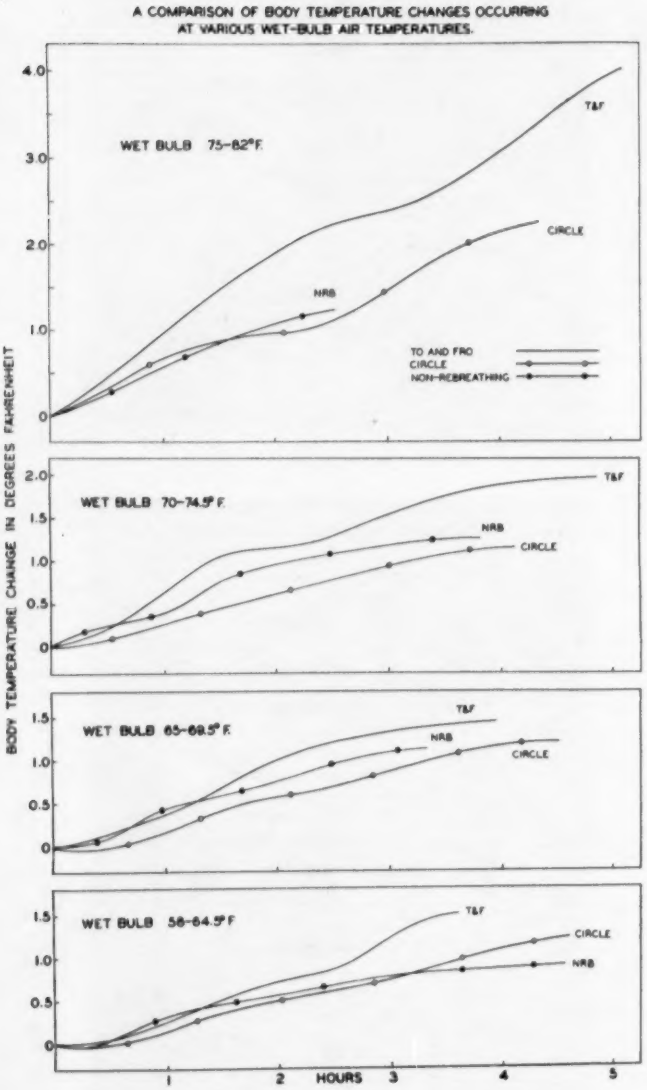

Fig. 5.—Temperature in three anesthesia systems.

Treatment methods considered in relation to their relative effectiveness are shown in Figure 2. The aim of treatment is the rapid reduction of the elevated body temperature. Permitting body temperature to remain markedly elevated is to risk circulatory collapse and injury to the central nervous system. The most effective methods of treating pyrexia during surgery are those which utilize conduction loss—ice bags and the water-cooled mattress. The effectiveness of ice bags properly placed and used in sufficient number is clearly shown in Figure 3. Evaporating liquids on the skin has been found ineffectual because of the limited surface area the surgical drapes allow for application of these substances.

The importance of heat loss through evaporation of sweat has been mentioned. The key relationship upon which heat loss by evaporation depends is the difference between the vapor pressure of water at skin temperature and the vapor pressure of water in room air. A close parallelism exists between the latter and the wet-bulb temperature as determined by hygrometry. For these reasons the wet-bulb temperature is the most important factor in determining the ability to tolerate environment. Figure 4 shows the rectal temperature changes observed in a group of afebrile adult subjects during surgery, in relation to the wet-bulb temperature of the room. The trends of body-temperature rise follow intimately corresponding rises in wet-bulb temperature. Above 75 F. wet-bulb temperature this effect is marked and heat retention becomes a frequent complication.

Use of the to-and-fro anesthesia system, especially in a humid environment, is associated with significantly higher rises in body temperature than is the case when either circle absorption or nonrebreathing systems are used. The temperature observed in these three systems and in the lower trachea of anesthetized patients is shown in Figure 5. The heat of neutralization released in the soda-lime canister and the proximity of the canister to the patient in the to-and-fro system results in a gain in heat to the body of about 4% of the basal heat production when this system is used. Use of the circle or nonrebreathing systems results in a loss of about 4% and 16%, respectively. In the latter circumstance, nonrebreathing system, the heat loss is the result of evaporation of water from the lung to saturate the inspired dry gases.

Case Reports

IDIOPATHIC HEMORRHAGIC INFARCTION OF THE SMALL INTESTINE

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THE BASIS of this paper is a study of 12 cases of massive infarction of the small intestine consequent to mesenteric thrombosis. The infarct of the bowel was caused in each case by a thrombus which occluded the vein draining the infarcted area. The condition appeared rather suddenly in persons who were otherwise known to be healthy. Of the 12 patients treated, only 2 survived extensive resection of the bowel; the other 10 died. Of the two surviving patients, one was a 79-year-old woman and the other a 54-year-old man.

We wish to state at the outset that the present study is concerned only with primary mesenteric thrombosis and intestinal infarction. This article is not concerned with infarctions resulting from mechanical strangulation of the bowel produced by volvulus, kinks, or hernia, or from embolic occlusion of the mesenteric vessels.

A review of the surgical literature bearing on the subject gives the impression that primary mesenteric thrombosis and the concomitant intestinal infarction is a rare occurrence. This point is emphasized here in order to suggest the possibility that the condition is probably commoner than the literature would suggest and that owing to late diagnosis or to lack of appreciation of the seriousness of this condition many of these patients die without an accurate diagnosis having been established.

Of the 12 patients reported on in this paper, 9 were males and 3 were females, showing a preponderance of the disease among male patients. Although these figures are too small for accurate comparison, they suggest that the disease is much more prevalent in males. That the comparison may be a true one, however, is suggested by the fact that the figures compare favorably with other figures reported in the literature.

Mesenteric thrombosis may occur at any age but is most often seen in persons about middle life. The ages of the patients in this series ranged from 48 to 79 years, averaging 56 years. The age period of greatest frequency was from 48 to 55 years.

The onset of symptoms was insidious in eight cases; in two instances there was a history of vague abdominal pain for a period of two weeks preceding the acute onset. In the remaining four cases the onset of symptoms was acute and was described as sharp, gnawing, or cramp-like in character. The location of the pain varied and usually conformed with the segment of bowel that was involved. It appeared in most instances that the pain was much too severe to be accounted for by the physical signs. Nausea and vomiting was present in all 12 patients. In one instance the vomitus contained blood; in three others there was passage of blood through the rectum.

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Physical examination in all 12 patients revealed fairly well-developed and well-nourished persons who appeared quite ill; 3 patients were in a state of shock. The average temperature varied from 98.2 to 100 degrees F.; the pulse was rapid. In most instances the abdomen was distended and tympanitic; in one instance it was flat. Various degrees of tenderness were elicited in all of the patients. In four of the patients local rigidity of the abdominal wall was found and in two others, diffuse muscle spasm. In three patients rectal examination disclosed the presence of blood on the examining finger.

The white blood cell count was elevated in most cases and ranged between 18,000 and 32,000 per cubic millimeter. Patients who had lost considerable amounts of fluid by vomiting or diarrhea showed evidence of hemoconcentration.

The preoperative diagnosis was mesenteric thrombosis in two patients, acute pancreatitis in two, acute appendicitis in four, perforated carcinoma of the bowel in one, perforated diverticulum in one, coronary occlusion in two, and intestinal obstruction in one. These figures illustrate how difficult it is to make a correct diagnosis, since the clinical picture is so extremely variable.

At operation all 12 patients showed serosanguineous fluid in the peritoneal cavity and within the lumen of the affected bowel. There was a striking similarity in the pathological picture all 12 patients presented; it consisted of a hemorrhagic infarct involving a large segment of the small intestine. The size of the infarcted bowel varied and measured 120 to 300 cm. in length. In four instances almost the entire length of the small intestine was gangrenous. The infarcted intestine presented itself at operation as a purple-red, shining, and distended tubular structure with an edematous, soft, and friable wall that was well demarcated from the adjoining normal-looking intestine. The veins draining the infarcted bowel were prominent and showed macroscopic evidence of thrombosis.

Microscopic examination of the specimens again showed the same histological picture in all patients. It consisted of congestion and thrombosis of the venous channels and hemorrhagic extravasations into the bowel wall. The thrombi were relatively fresh and consisted largely of platelets, marginated white blood cells, and agglutinated erythrocytes. There was, in addition, a mild inflammatory reaction in which the exudate consisted, for the most part, of small and large monocytes and polymorphonuclear leucocytes. The various coats of the bowel wall were poorly delineated and in many instances only ghosts of preexisting tissue structures could be recognized.

CASE 1.—A 79-year-old white woman was admitted to the Jewish Hospital because of diffuse abdominal pain, nausea, vomiting, and constipation of 24 hours' duration. The pain at the onset was dull and intermittent but later became sharp and colic-like in character. There was no bowel evacuation during all this time, and enemas were ineffectual. There was no history of any previous abdominal complaint, operation, or injury.

On physical examination the patient appeared as a frail old woman, acutely ill, and in a state of semishock. There was a very pronounced abdominal distention but very little tenderness or rigidity. The temperature was normal, pulse rapid, and blood pressure 100/90. The blood count revealed 23,000 white blood cells, 80% of which were polymorphonuclear leucocytes. A flat x-ray film of the abdomen showed a step-ladder pattern of the small intestine with evidence of gaseous distention and well-demarcated fluid levels.

The clinical impression was that of intestinal obstruction of undetermined origin. At operation, performed by one of us (J. R.), the peritoneal cavity contained a large amount of serosanguineous fluid. About 7 ft. (213 cm.) of small intestine was found infarcted and of a dark

purple-red color. In addition, the wall was edematous, shiny, and friable. The bowel was very distended and contained a large quantity of dark hemorrhagic fluid within its lumen. The mesenteric vessels draining the infarcted intestine were prominent and thrombosed.

The entire segment of the infarcted intestine, including a few centimeters of adjoining normal bowel on either side, was resected together with a portion of attached mesentery. The operation was terminated with a side-to-side anastomosis of the opposed ends of the severed intestine.

Grossly, the resected intestine was the seat of a hemorrhagic infarct and measured 215 cm. in length. The wall was of a dark purple-red color, very shiny, and friable. The lumen contained a large amount of hemorrhagic fluid. The corresponding mesenteric vessels were occluded by fresh thrombi. Microscopic examination of the specimen revealed an infarct of the bowel caused by thrombotic occlusion of its venous supply.

After the operation the patient was placed in an oxygen tent for 24 hours, was given a blood transfusion, and was fed parenterally during the first 48 hours. The bowel was kept decompressed by means of continuous Wangensteen suction. She made an uneventful recovery and was discharged from the hospital on the seventh postoperative day. At the time of writing, almost five years since the operation, the patient is alive and well.

Although the patient was critically ill and her condition so grave that a fatal outcome was expected, it was nevertheless felt that surgery offered the only chance of survival; it was clearly shown that failure to operate will unquestionably lead to a fatal outcome. The patient in this case survived the operation in spite of the extensive procedure, her old age, and her extremely weakened physical condition.

Case 2.—A 54-year-old man was admitted to the Mount Royal Hospital with the complaint of abdominal pain, nausea, and vomiting of seven days' duration. At the onset he had vague digestive disturbances consisting of indigestion, nausea, and belching. The patient then noted a gradual increase in pain which became steady and gnawing in character. This was associated with almost continuous vomiting during the previous 12 hours. His past history was irrelevant; there was no history of trauma or previous abdominal surgery.

On examination the patient was found to be a well-developed and well-nourished man who appeared acutely ill. The temperature was 100 F., pulse rate 110, respiration rate 24, and blood pressure 115/80. The abdomen was diffusely tender, not rigid but very much distended. The blood count showed 25,000 white blood cells, 78% of which were polymorphonuclear leucocytes.

The clinical impression was that of ruptured appendicitis and peritonitis. At operation performed by one of us (P. R.) through a right rectus incision the peritoneum was opened and a large quantity of free serosanguineous fluid was found in the peritoneal cavity. About 5 ft. (152 cm.) of jejunum and ileum were infarcted and resected, followed by a side-to-side anastomosis. The patient ran a stormy course during the first few days postoperatively, after which he began to improve and was discharged from the hospital 10 days later.

Pathological examination of the specimen showed 150 cm. of necrotic bowel. The latter was edematous, deep purple-red in color, shiny, and friable. Microscopically, the bowel showed hemorrhagic extravasation into the wall and multiple thrombotic occlusions of the small venous channels.

This case also illustrates the fact that surgery should be undertaken even in the extremely ill patient. No other possible chance of life can be offered to the patient unless he is operated upon and the gangrenous bowel removed.

EVALUATION

The symptoms of intestinal infarction are very variable and cannot be said to conform to any definite type. Correct diagnosis is therefore unusual. It has been said, indeed, that the picture presented forms a composite of intestinal obstruction and internal hemorrhage.

Perhaps the most constant feature of the disease is acute abdominal pain and distention, frequently associated with nausea and vomiting. The pain may be sharp, colicky, or gnawing in character. Another striking symptom in the syndrome of

this disease is that early in the attack the pain may seem to be out of all proportion to the meager abdominal findings. The abdomen is usually widely and definitely tender to palpation, and the abdominal rigidity is not nearly as marked as one might expect it to be when the possible duration of the complaint and tenderness are considered. The patient complains of a severe, constant, gnawing abdominal pain centering mainly in the midportion of the abdomen. Passage of blood by rectum is of considerable diagnostic significance.

Physical examination is usually inconclusive. The abdomen is, for the most part, distended and tympanitic although it may be flat and dull to percussion. Rectal examination may disclose the presence of blood on the examining finger, a most significant diagnostic aid which may lead one to suspect the diagnosis of mesenteric thrombosis. The normal leucocytic relationship is very much disturbed, and the white cell count may reach very high figures. The temperature range in the uncomplicated picture is low or only slightly elevated. Roentgenologic examination of the abdomen may show gaseous distention with fluid levels suggestive of intestinal obstruction. Unless the possibility of this condition is kept in mind it will be very difficult to recognize it as a definite surgical problem, although surgery offers the only chance of life. It may be generally postulated without fear of criticism that if after thorough study of the history, physical findings, and results of laboratory and x-ray investigations the diagnosis is still obscure then mesenteric vascular occlusion should be considered as a most likely diagnosis. To prove this thesis, however, one must exclude from diagnostic consideration any other intra- or extra-abdominal condition which may mimic this lesion. If there were a more accurate way of determining the diagnosis, one of the worst obstacles to the management of these cases would be eliminated. It may be also added that the diagnosis of mesenteric thrombosis would become more certain if fresh blood is passed by rectum or if digital examination of the rectum discloses the presence of blood on the examining finger.

Most cases of mesenteric thrombosis that we encounted are arterial in type and are caused by emboli; the venous type is uncommon and little is known about its etiology, but what better field could there be for speculative thought?

Many and varied suggestions have been offered to explain the etiology and pathogenesis of mesenteric thrombosis. All investigators agree that the infarction of tissues follows strictly in the path of, and is confined to, the thrombosed vascular area. In regard to the immediate mechanism of this thrombosis, there is no unanimity of opinion. In a general way it may be said that more than one causative factor may be responsible for the production of local thrombosis in individual cases. It would be of interest to enumerate in this connection the various conditions under which local thrombosis may develop in the mesenteric veins.

Venous thrombosis following mechanical strangulation by volvulus, hernia, or kinks of the bowel is a well-recognized entity. The condition is caused by a twist or kink of the bowel or constriction of a loop of intestine by a tight hernia ring that results in acute circulatory disturbance. If the twist, kink, or hernial constriction is not relieved immediately, the structures distal to the lesion become gangrenous and irreversible damage may be done to the tissues. This explanation of mesenteric thrombosis, as it concerns this series of cases, is open to question. None of the patients in this series showed at operation any evidence of mechanical strangulation of the bowel.

Thrombi may be liberated from some distant point in the general circulation and later become arrested in a mesenteric vessel as an embolus. As already stated, most cases of mesenteric occlusions that we encounter are arterial in type and are caused by emboli. The venous form of mesenteric occlusion is rarely, if ever, embolic in nature. In order for such a condition to occur one must assume the existence of an arteriovenous aneurysm of the mesenteric vessels and such a condition is most unlikely. Moreover, there was no evidence in any of the cases here cited of a lesion existing elsewhere in the body from which emboli might become detached.

We are of the opinion that trauma to the mesenteric veins plays an important role in the development of local thrombosis. Our contention is well supported by animal experimentation. It is a well-known physiologic principle that injury to a blood vessel will initiate local clot formation. In a series of experiments conducted by one of us [1] it was shown that a forceful pull on the jugular vein in the rabbit, which injured its endothelial lining, resulted in the formation of a clot at the site of injury. Furthermore, if the lumen of the vein distal to the point of injury was partially constricted so as to retard the flow of blood through the vein, thrombosis occurred more promptly. There was a striking resemblance in the character of the thrombus thus produced experimentally to that seen in the thrombosed mesenteric veins. Both types of thrombi were composed of platelets, marginated leucocytes, and agglutinated red blood cells. In the light of such experimental findings, it is our belief that the mechanism involved in the formation of mesenteric thrombi and that which is produced experimentally in animals is the same. We believe that a possible stretching of a mesenteric vessel may occur consequent to some trauma, and this in turn leads to the formation of a thrombus. It would seem then that trauma must be envisaged as a likely possibility in the causation of mesenteric venous thrombosis.

It is also of interest to note in this connection that the mesenteric veins become engorged with blood during the process of digestion. This is of significance because a temporary retardation in the flow of blood through the mesenteric venous circulation occurs during the process of digestion. An injury to the endothelial lining of the vessel during this phase is likely to incite local clotting within the mesenteric veins. Such a conclusion is also in harmony with the experimental findings reported by one of us (J. R.), of which mention has already been made.

From the foregoing analysis it would seem justifiable to conclude that trauma to the mesenteric veins is an important etiologic factor in the formation of a clot. Such a theory would receive added support if there was a history of trauma antedating the onset of symptoms. So far as the present series is concerned, no such history of trauma was obtainable. It must be observed, however, that the absence of a positive history of an injury does not necessarily preclude its occurrence. In proof thereof is the fact that instances of intra-abdominal trauma have been observed in patients who at no time were aware that an injury had ever occurred. One of us (J. R.) has recently observed a case of a woman who presented clinical signs of acute intestinal obstruction and at operation was found to have a tear of recent origin in the broad ligament. Herniating through this rent in the broad ligament was a strangulated loop of intestine. When the patient was subsequently asked whether she was injured before she was taken ill, she denied such knowledge, yet it was quite obvious that the tear in the broad ligament was traumatic in origin. Evidently, then, a patient may sustain an injury and yet not be cognizant of its occurrence.

It is not our contention that all cases of mesenteric thrombosis arise in the manner we have indicated. There is no physiologic feature to refute the thesis that local clot formation in a mesenteric vessel may develop in some other way. It seems reasonable, however, to conclude that in a very significant number of cases mesenteric venous thrombosis is a result of trauma to the vein.

Our clinical and experimental observations may be summed up by saying that trauma is an important factor in the production of mesenteric venous thrombosis. This is particularly true in cases in which no other palpable reason can be found to account for the clot formation.

Periarteritis nodosa as a cause of mesenteric occlusion and intestinal infarction is well recognized, although the condition is very uncommon. One of us (J. R.) has observed a case of massive infarction of the bowel (to be reported later) caused by periarteritis of the mesenteric vessels. This peculiar inflammatory lesion involves primarily the small arterioles and is therefore outside the scope of the present paper.

Surgeons wish to know, from experience gained from proved results, what the most effective treatment is for a given condition. Owing to the difficulty in early diagnosis in this obscure type of lesion, very few patients have been known to survive operation. Diagnosis is invariably late, as it is dependent on the onset of symptoms, and it is well recognized that unfortunately the early symptoms of this disease are vague and are likely to be confused with those of other intra-abdominal lesions. By the time the patient sees the surgeon the disease has reached the terminal stages. It seems that modern surgery has not altered this gloomy prognosis, especially when the disease is allowed to advance to a stage where the changes in the bowel have become irreversible.

Once the diagnosis of mesenteric vascular occlusion has been made, operation should be performed at once, even in the critically ill patient. Our studies lend full support to the contention that failure to operate early will lead to a fatal termination in almost every case. Therefore the longer the interval between the onset of symptoms and operation, the worse the prognosis. The choice of operative procedure depends on the extent of infarction and the condition of the patient. The ideal treatment is resection of the entire segment of infarcted bowel, including a few centimeters of adjoining normal-looking bowel. The continuity of the bowel, whenever possible, should be restored at once, preferably through a side-to-side anastomosis. The resection can be done quickly without undue strain on the patient. Heparin may be given immediately after the operation for the first few days and then be gradually replaced by bishydroxycoumarin (Dicumarol) in sufficient quantities to keep the prothrombin time around 35 seconds. Supportive treatment consisting of blood transfusions, parenteral administration of fluids to keep water and electrolyte balance, antibiotics, etc., should be given as required in the individual case.

SUMMARY

A review has been presented of 12 cases of mesenteric venous thrombosis and intestinal infarction. In two patients the infarcted bowel was resected successfully. In the remaining 10 cases the lesion was too far advanced at the time of operation and resulted therefore in death in each instance.

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DUPLICATION OF TWO PARTS OF THE ALIMENTARY TRACT

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IN THIS discussion the term "duplication of the alimentary tract" will have the connotation applied by Ladd and Gross.[1] They use it to refer to any cyst or diverticulum associated with the alimentary tract (except diverticula of the Meckel variety), the wall of which contains smooth muscle and is lined with a mucosa capable of having been derived from entoderm. Where the duplication is in the form of a diverticulum, it differs from the acquired variety in that the muscle in the wall is common to both the diverticulum and the adjacent bowel.

Duplication of the Alimentary Tract

Situation	Cases, No.
Base of the tongue	1
Intrathoracic	61
Connected with stomach	6
Connected with duodenum	22
Connected with small intestine (chiefly terminal ileum)	136
Connected with ileocecal junction	21
Connected with cecum	17
Connected with remaining colon	9
Connected with rectum	10
Intraperitoneal but not connected with alimentary tract	8
Retroperitoneal but not connected with alimentary tract	4
Preperitoneal (in relation to umbilicus)	20
Total	315

Duplications are by no means rare. Dohn and Povlsen,[2] in a recent review of the literature, found reports on 315 cases. The duplicated portions were distributed along the entire length of the alimentary tract, as is shown in the Table. Multiple duplications were present in 13 cases. When the duplication occurred in the abdominal cavity, the mucosa usually corresponded in type to that of the adjacent bowel, but in 20% of the cases the pattern was that of some entirely different segment of the alimentary tract. In 58 intrathoracic duplications, the lining was composed of the following types of epithelium: gastric in 23, columnar ciliated in 15, stratified squamous in 3, intestinal in 4, and mixed in 13. Olenik and Tandatnick,[3]

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From the Section of Surgery (Dr. Hallenbeck) and the Section of Pediatrics, Mayo Clinic (Dr. DuShane), and from the Ochsner Clinic (Dr. Ochsner).

in a review of 74 cases of duplication in the mediastinum, found that the type of the lining epithelium was respiratory in 35 cases, primitive esophageal in 12, gastric in 15, intestinal in 4, and mixed in 8.

CLINICAL FEATURES

Dohn and Povlsen [2] were able to trace the clinical histories in 124 cases of intra-abdominal duplication. Obstruction of the bowel was the presenting feature in 83 cases. The obstruction usually was due to compression of the bowel, but in 12 of the 83 cases the duplication formed the point of origin of an intussusception, and in 9 cases it was responsible for volvulus of the small intestine. Pain was the presenting feature in 22 cases, and in half of them the pain was intermittent. The intermittency could be due to recurring intussusception or volvulus, or to alternating pressure in a cyst as a result of a varying rate of secretion from its wall, or to alternating contraction and relaxation of the wall. In 7 cases the only symptom of note was the presence of a mass; in 60 cases a mass was palpable. In four instances the picture was that of peritonitis due to pressure atrophy of the intestinal wall. The presenting sign in two cases of duplication of the stomach was fistula.

In the thorax the clinical picture tends to be of a more chronic nature. Dohn and Povlsen [2] noted that 14 of 41 patients were free of symptoms, and of the remaining 27, more than half presented symptoms in the first year of life. The symptoms were those of pressure on the respiratory tract—cough, expectoration, stridor, and dyspnea—more rarely, those of pressure on the alimentary tract. Gastrogenic cysts on occasion ruptured into a bronchus or lung, giving rise to severe hemoptysis. Bleeding also resulted from mere pressure on the bronchial tree or esophagus. Roentgenograms were invaluable in locating these tumors, which usually were in the posterior mediastinum.

TREATMENT

Abdominal duplications must usually be excised together with the adjacent portions of the bowel. Gardner and Hart [4] reported internal drainage of a cyst of the duodenum, accomplished by anastomosing the cyst and the duodenum.

Intrathoracic duplications occur in relation to the trachea and bronchi or to the esophagus and usually can be excised.

REPORT OF A CASE

A girl, 11 months old, was seen at the Ochsner Clinic in December, 1951, after the presence of an intrathoracic tumor had been discovered in a roentgenogram. A rounded lesion occupied the left upper pulmonary field and displaced the posterior mediastinum to the right. In addition, the sixth thoracic vertebra was a hemivertebra, and the lateral masses of the fourth had not fused. On Dec. 20, the left hemithorax was explored (Ochsner) and a cystic tumor, 4 cm. long and 2.5 cm. in diameter, was removed from the posterior mediastinum. For several months the child's condition progressed satisfactorily, and she gained weight.

On June 9, 1952, however, the hemoglobin value was found to be 44%. Results of other studies of the blood were within normal limits, and repeated examination of stools at that time failed to reveal evidence of blood. A blood transfusion was given by her home physicians.

On Sept. 13 the child had a tarry stool, and a further transfusion was given. On Sept. 26 the value for hemoglobin again was 45%, and again this condition was remedied by transfusion.

The child was brought to the Mayo Clinic on Sept. 30. She was rather listless and had typical mongoloid features. There were no other noteworthy features, apart from some prominence of the abdomen.

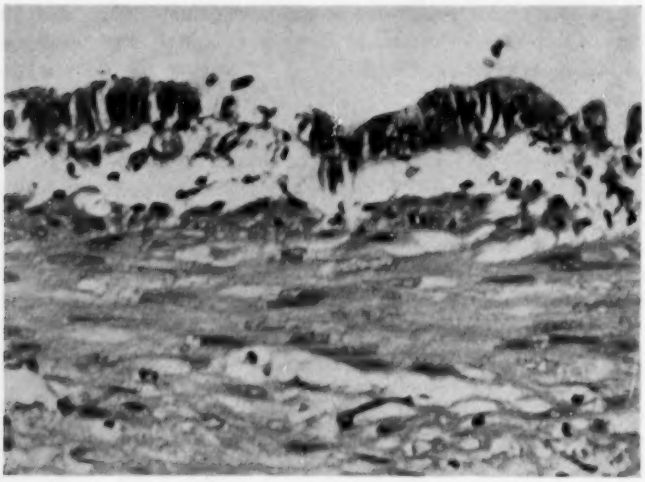

Fig. 1.—Section through wall of intrathoracic cyst. Lining is columnar epithelium. ×415.

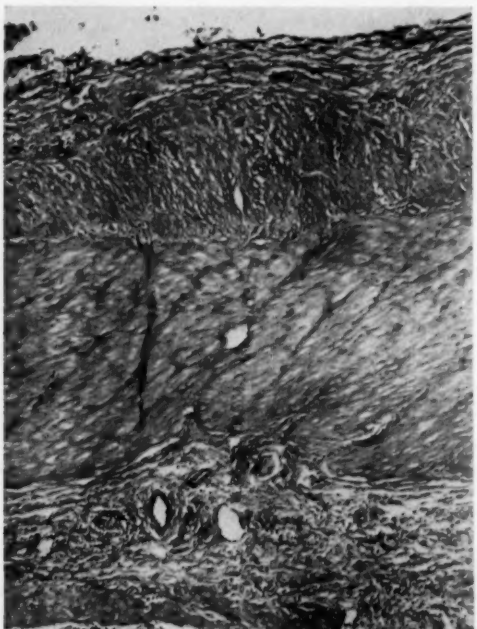

Fig. 2.—Section through wall of intrathoracic cyst, showing muscularis mucosa, a submucosal layer, and then two distinct layers of muscle. On higher magnification the cells between these two layers were definitely nerve cells. ×65.

Laboratory investigation revealed a concentration of hemoglobin of 13.1 gm. per 100 cc. of blood. Erythrocytes numbered 4,360,000; leucocytes, 11,600, and platelets, 164,000, all per cubic millimeter of blood. The differential blood count was within normal limits. A blood smear revealed moderate anisocytosis, hypochromasia, and polychromasia. The platelets appeared morphologically adequate. The coagulation time was six minutes. Urinalysis gave negative results, but guaiac and benzidine tests of the stool indicated the presence of occult blood. Roentgenograms of the thorax confirmed the presence of the vertebral abnormalities noted above. Those of the esophagus, stomach, duodenum, and colon did not give evidence of a pathologic process. Proctoscopic examination gave negative results.

It was believed that the melena might be caused by a lesion of the small intestine, such as a Meckel diverticulum. On Oct. 10, therefore, exploration was carried out, and an inflamma-

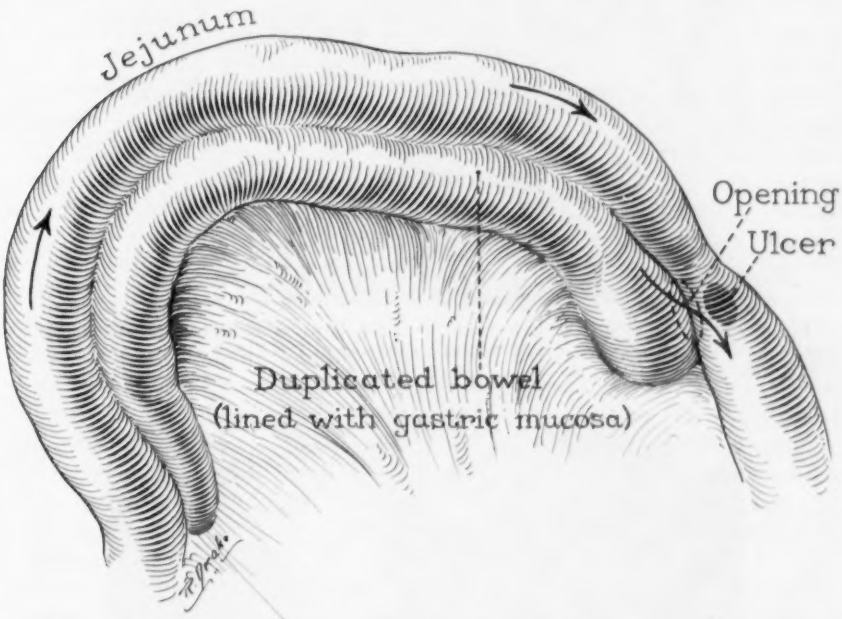

Fig. 3.—Drawing of the duplicated bowel and the jejunum, showing the position of the ulcer.

tory mass, the result of a penetrating ulcer, was found in the jejunum. On the mesenteric side of the jejunum, within the mesentery, there was a duplicated portion of intestine approximately 8 in. (20 cm.) long. The segment of jejunum, together with the duplication, was excised, and end-to-end anastomosis restored continuity.

The child had an easy convalescence and was dismissed on the 13th postoperative day.

Pathology.—The pulmonary cyst measured 4 cm. in length and 2.5 cm. in diameter. It had a ragged, fibrous, external capsule, varying in color from light pink to red. The wall was 2 mm. thick, and the lining was smooth and shiny. The fluid inside the cyst was cloudy and yellow. The lining epithelium had largely disappeared, but where it was present it consisted of a single layer of columnar nonciliated epithelium (Fig. 1). Beneath this there was a thin layer of muscle compatible with the muscularis mucosa of the bowel. The fibers of two further layers of muscle ran at right angles to one another. Between these layers were ganglia and nerve fibers such as are seen in the myenteric plexus of the bowel wall (Fig. 2). The appearance was compatible with the diagnosis of duplication of the alimentary tract.

Another specimen consisted of a portion of jejunum 47 cm. in length (Fig. 3). Along the mesenteric border of the jejunum there was another structure 23 cm. in length, in the form of a hollow tube, closed at one end, and opening at the distal end into the jejunum. The jejunum and this structure were closely apposed, and no line of cleavage could be developed

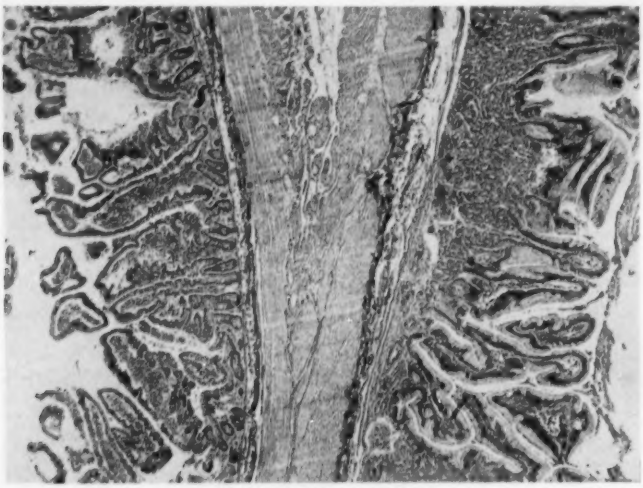

Fig. 4.—Section through the wall common to the jejunum and the duplication. Gastric mucosa is on the one side and jejunal mucosa on the other. The muscle of the respective walls fuses to form a common layer. ×22.

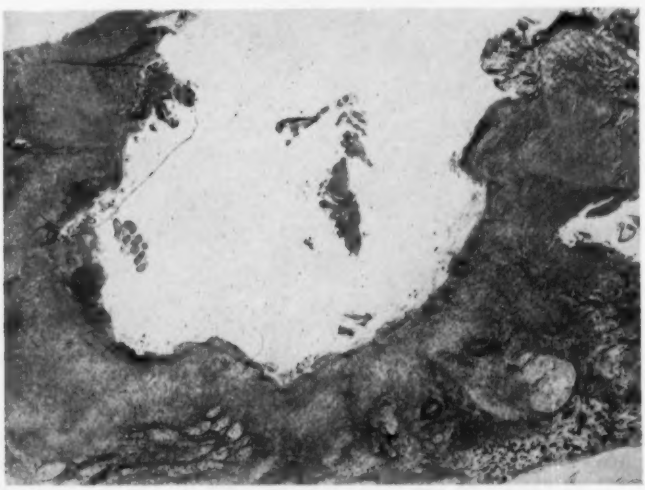

Fig. 5.—Complete penetration of the ulcer through the muscular coat of the jejunum. ×6.

between them. The duplicated portion tapered, measuring 2 cm. in circumference at the proximal end and expanding to 6 cm. at the distal end. When this portion was opened, the mucosa was seen to have prominent folds running longitudinally, consistent with the appearance of normal gastric mucosa and in contrast to the finer circular folds of the adjacent jejunum. Just

proximal to the opening of the duplicated portion the lumen of the jejunum was considerably narrowed, measuring 1 cm. in diameter. Furthermore, this narrowing formed the proximal border of an ulcer which was found in the wall of the jejunum just opposite the opening of the duplicated portion. The ulcer measured 1.5 cm. in diameter and was 0.5 cm. in depth. An omental pad of fat was adherent on the serosal surface of the jejunum just opposite the ulcer.

A section was taken through the wall common to both the jejunum and the duplication. On the one side was typical jejunal mucosa, but on the side of the duplication the mucosa contained chief and parietal cells and had the typical appearance of gastric mucosa. Between these layers of mucosa the muscle layers fused (Fig. 4). Another section demonstrated that the ulcer had completely penetrated the wall of the jejunum, destroying the muscle layers (Fig. 5).

COMMENT

Simultaneous occurrence of duplications in the thorax and in the abdomen is rare but has been noted on several occasions previously. Roth [5] reported the case of an infant who died just after birth. Necropsy revealed a mediastinal, a retroperitoneal, and a mesenteric cyst, all lined with intestinal mucosa. Roth [5] referred to similar cases reported by Roesfield and Hennig. Nicholls,[6] quoting Brass, mentioned additional cases of two children who died soon after birth; each had a cyst in the mediastinum and another in the abdomen, each lined with intestinal mucosa. Black and Benjamin [7] reported the case of a boy, 4½ months of age, who had repeated episodes of melena and finally died in shock. At necropsy he was found to have a retromediastinal enterogenous cyst, the lining of which could not be identified, together with a duplication alongside the ileum. Just at the place where this duplicated portion opened into the jejunum, and actually in the wall of the duplicated portion, there was a perforated peptic ulcer. The lining mucosa appeared to be jejunal in type. Poncher and Milles [8] reported the case of a boy, 2½ years of age, who had a history of bloody stools and attacks of cramping abdominal pain. On roentgenologic examination he was found to have a mediastinal cyst and this was removed. The patient died. Necropsy revealed an additional mediastinal cyst, also lined with gastric mucosa, and duplication of the ileum. The duplicated portion contained a gastric polyp and was lined with gastric mucosa. Opposite the communication into the ileum there was a peptic ulcer, presumably the source of the bleeding. As far as we know, our report, herewith, represents the first instance of successful removal of separate duplications, one from the thorax and the other from the abdomen.

Hemorrhage from the bowel rarely is the presenting symptom of duplication. The similarity of the patient of Poncher and Milles [8] to our patient is of interest. In Black and Benjamin's [7] case the ulcer was actually in the duplicated portion. Fisher [9] reported successful removal of a duplicated pouch which was lined with gastric mucosa and extended from the right hemithorax, through the retroperitoneal tissues, to open into the terminal portion of the ileum. There was an ulcer in the wall of the ileum opposite the opening, and the presenting symptom was hemorrhage. Sparrow * described the case of a girl, 8 years of age, who had duplication of the jejunum which was lined with gastric mucosa. Here the ulcer accounting for the bleeding was in the blind end of the pouch.

* Sparrow, T. D., in discussion on McCutchen, G. T.: Reduplication of the Stomach, Ann. Surg. **129**:830-831 (June) 1949.

SUMMARY

In the case reported the two duplications of the alimentary tract were respectively in the thorax and in the abdomen. Another unusual feature was hemorrhage from a peptic ulcer of the jejunum which caused considerable narrowing of the adjacent intestine. As far as we know, this is the first time that separate duplications, one in the abdominal and the other in the thoracic cavity, have been removed successfully from the same patient.

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SIMULTANEOUS TRIPLE PRIMARY GASTROINTESTINAL CARCINOMA

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THE STUDY of cancer including its diagnosis and treatment appears to be reaching its most intensive phase currently. Familial and hereditary predispositions, environmental influences of many kinds, racial tendencies, and other factors are all under profound scrutiny now in the battle against cancer. Of great interest and possibly of more than mere statistical value are the cases of multiple malignant lesions occurring in the same person. It is well known to the oncologist that, in general, those patients having developed one malignant lesion will have an increased tendency to subsequent affliction by additional primary cancers.[6] However, a breakdown of these large series of cases presenting multiple tumors, as has recently been reported by Mider and his associates,[1] reveals that this so-called increased cancer susceptibility is probably limited to only a few sites, for example, colon and female breast. Even in the groups of colon and breast cancer patients, this increased susceptibility remains confined to the identical tissue, i. e., remaining colon * or opposite breast. Actually, Mider and his co-workers [1] conclude that there was no increased liability to the development of a second cancer in an organ or tissue different from that in which the primary tumor occurred (where the primary cancer occurred in the hematopoietic system, uterus, stomach, breast, or large intestine). Thus, subsequently or simultaneously occurring multicentric cancers arising in different organs in the same patient remain somewhat unusual and certainly as unpredictable as any new growth.

Pemberton and Shands [5] stated that the involvement of colon and stomach by multiple primary malignant growths, either simultaneously or subsequently, was rare. They collected from the literature a total of 51 cases of primary carcinomas of the stomach and colon occurring in the same patient. In 41 of these cases the tumors were simultaneous. Bacon and Tavenner,[3] in reporting 94 cases of multiple primary malignant lesions involving colon or rectum, found only 3 cases in which the primary tumors occurred in the stomach and large intestine.

Concerning treatment of these coexistent tumors, Pemberton and Shands [5] reported, in 1952, the second successful surgical removal of simultaneously occurring primary carcinomas of the stomach and colon at one operation. This would certainly be the surgery of choice, provided, of course, that both lesions are operable and that the patient's condition is satisfactory.

I should like to present a case which is both rare and rather interesting. Three simultaneous primary carcinomas were found in this man (stomach, cecum, rectum). I believe this is the first such case reported. In addition, a simultaneous right colectomy and subtotal gastrectomy were done as the first operative procedure to efface two of these tumors. This is the third such instance recorded of simultaneous removal of primary carcinomas of both colon and stomach.

From the Department of Surgery, Baylor University Hospital.

* References 2, 3, and 4.

REPORT OF A CASE

W. H. H., a 64-year-old white man, was admitted to the Baylor University Hospital Surgical Service on July 10, 1951, complaining of abdominal pain and rectal bleeding of approximately four months' duration.

The family history was negative for cancer, and the past history revealed only that the patient had had mild Parkinsonism for the past 10 years.

Present Illness.—Approximately four months prior to admission the patient noted the passage of small amounts of bright red blood per rectum with each bowel movement. Three months prior to admission he noted a gnawing-type abdominal pain which he was unable to localize to any one point in the abdomen. During this period he also noted mild anorexia and

Fig. 1.—Photomicrograph of gastric tumor reveals typical mucoid or colloid type carcinoma composed largely of irregular lakes of mucin containing numerous signet-ring cells and clusters of relatively undifferentiated tumor cells. A small area of gastric mucosa which is distorted by inflammation is seen at upper right (approximately × 75).

some generalized weakness and had lost 7 lb. (3.2 kg.). There had been no change in bowel habits except for mild constipation in the past few months.

Physical Examination.—The physical examination was essentially negative, except for the rectal examination which disclosed a soft polypoid lesion on the posterolateral aspect of the rectum approximately 8 cm. from the anal orifice. This lesion was approximately 3 cm. in diameter and had a sessile base. Proctosigmoidoscopy revealed a polypoid cauliflower-type soft lesion on the posterolateral mucous membrane of the rectum, which was reddish-gray in color, friable, and bled readily. A biopsy specimen of the lesion was reported as adenocarcinoma.

Laboratory Studies.—A chronic blood-loss type anemia was noted, the hemoglobin being 7.6 gm. per 100 ml. of blood. Numerous other hematological studies were within normal limits. Several stool examinations revealed the presence of occult blood. An upper gastrointestinal series revealed no evidence of disease in the esophagus, stomach, or duodenum. Chest x-rays

revealed no evidence of intrathoracic disease except for moderate pulmonary emphysema and moderate hypertrophic arthritis of the thoracic vertebra. A barium enema revealed no evidence of disease of the colon above the rectum.

Hospital Course.—After routine preoperative preparation, including sterilization of the colon with antimicrobials and several transfusions of whole blood, the patient underwent exploration on July 21, 1951, for the purpose of doing an abdominoperineal resection for the adenocarcinoma of the rectum.

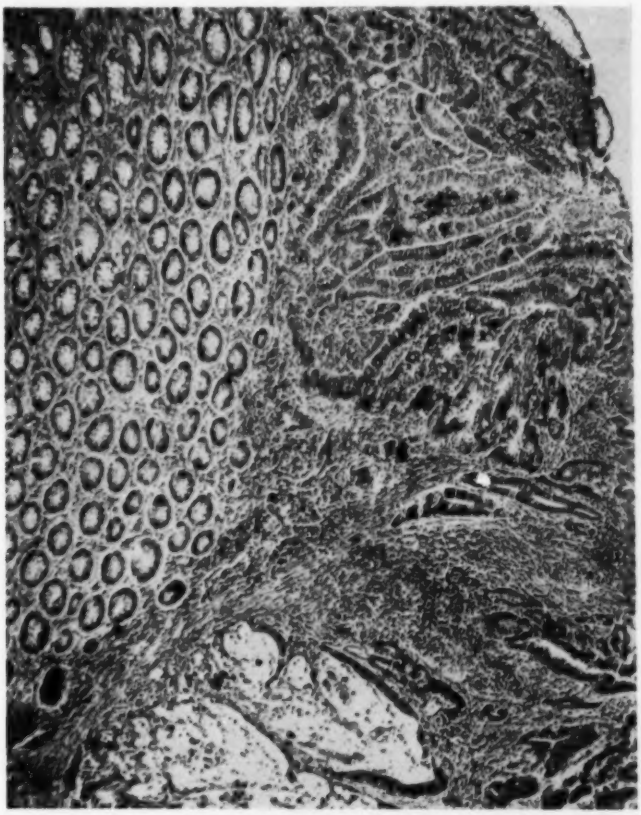

Fig. 2.—Area at edge of cecal lesion reveals a definite transition from normal mucosa to carcinoma. Tumor is composed largely of atypical but definite gland-like structures with individual cell pleomorphism, nuclear irregularity and hyperchromatism, loss of polarity, abundant abnormal mitoses, and the other classic features of anaplasia (approximately × 75).

The abdomen was entered through a left paramedian incision, and a thorough visceral exploration revealed, in addition to the rectal tumor, an umbilicated hard tumor mass about the size of a large hen egg within the cecum just inferior to the ileocecal valve and a third similar tumor on the lesser curvature of the stomach 4 cm. proximal to the pylorus. The remaining viscera were entirely normal grossly, as were the areas of lymphatic drainage of all three tumors. Since these tumors appeared grossly operable, a radical subtotal gastrectomy with an isoperistaltic retrocolic gastrojejunostomy (Hofmeister type) and a radical right hemicolectomy were done. The wound was closed in routine fashion with nonabsorbable surgical sutures, and the patient was returned to his room in good condition, having received 1,500 cc. of blood intravenously during the procedure.

Convalescence following this surgery was uneventful, and, on Aug. 3, 1951, the bowel having been sterilized again, a classic abdominoperineal proctosigmoidectomy was accomplished. He had withstood the surgery well and had received 1,000 cc. of blood intravenously during the procedure.

Convalescence following the second operative procedure was marred by an immediate postoperative hemorrhage from the perineal wound, which was remedied by religation of the right inferior hemorrhoidal artery, and by a sudden massive right-sided atelectasis occurring 24 hours postoperatively, this being corrected promptly by bronchoscopy. He thereafter had a completely

Fig. 3.—Zone of transition from normal mucosa to a classic relatively well-differentiated adenocarcinoma is clearly seen in this tissue section taken at the edge of the rectal growth (approximately × 75).

uneventful convalescence, being discharged from the hospital on Aug. 21, 1951, to be followed in the outpatient clinic.

The perineal wound was completely healed and the colostomy appeared quite satisfactory and was well regulated six weeks postoperatively. Fourteen months after the initial operation, in September, 1952, a complete laboratory evaluation and physical examination failed to reveal any evidence of new, recurrent, or metastatic tumor. At the time of this writing, 25 months following surgery, the patient remains at home with no abdominal complaints whatever but persistently refuses to return to the clinic for further study. As far as can be determined by telephone conversations with the patient and his close relatives, he still remains apparently free of tumor.

Pathological Report.—The lesion within the stomach was an ulcerated fungating tumor with everted edges and measured 4 by 1.8 cm. The tumor mass in the cecum measured approximately 4 by 1.3 cm. It was ulcerated similarly and had everted, piled-up edges. The third tumor, which was a fungating papilloid mass measuring 3.5 cm. in diameter, was found 8.5 cm. from the ano-rectal junction.

Histological examination of the gastric tumor revealed it to be a mucoid carcinoma, which nowhere showed the fairly definite glandular structures seen in the large bowel tumors (Fig. 1). Both the cecal and rectal lesions were seen to be typical adenocarcinomas with only the cecal tumor showing, in addition, an occasional area of so-called mucinous degeneration (Figs. 2 and 3). Careful microscopic scrutiny of all lymph nodes from each of the three specimens failed to disclose any evidence of metastasis.

COMMENT

The establishment of these three tumors as separate primary carcinomas can be accomplished in accordance with the widely accepted criteria of Warren and Gates.[6] These criteria are as follows: 1. Each of the tumors must present a definite picture of malignancy. 2. Each must be distinct. 3. The probability of one being a metastasis of the other must be excluded. The older criteria of Billroth have been almost completely abandoned today † because of their impracticable restrictions. First, all three tumors are unequivocally carcinomas. The gastric lesion is a classic mucoid or colloid type carcinoma. The remaining tumors are the usual adenocarcinomas. Second, all three neoplasms were separate and discrete, being widely removed anatomically, one from the other. Finally, the probability of one representing a metastasis of the other can fairly well be ruled out. The gastric tumor is histologically different from the other lesions. This would leave the cecal and rectal growths to be discussed. There were no metastases, either grossly or microscopically, from either tumor to peritoneum, liver or other viscera, omentum, mesentery, or regional nodes. A chest x-ray was also normal. It appears grossly illogical to consider a rectal lesion giving rise to a solitary metastasis of equal size in the mucosa of the cecum without setting up widespread metastases of similar size elsewhere, since this most certainly should be blood-borne spread, all lymph nodes and channels being negative microscopically; the reverse is likewise stated for the cecal growth. Microscopic examination at the edges of both of these tumors, in addition, revealed a classic transformation from normal mucosa to carcinoma (Figs. 2 and 3), this being profound evidence for each of them being primary new growths. The patient was also perfectly well and free of tumor, as determined by a thorough physical and laboratory examination 14 months postoperatively, and is now known to remain well 25 months following surgery, with further laboratory evaluation being refused. It is improbable to assume that the only blood-borne metastasis from either the cecal or rectal carcinomas had been removed. It seems quite likely that if either the rectal or cecal lesion were a true metastasis from the other, still further metastases would probably have made their presence known after an interval of 25 months.

Metastasis of gastric carcinoma to the rectal submucosa has been reported,[7] but in these cases there is evidence usually of dissemination elsewhere by the tumor, large primary lesions in the stomach, lack of involvement of the rectal mucosa itself by the metastasis, and histologic identification of the metastasis with the primary tumor.

† References 1 and 3.

Of additional interest in the case presented, was the failure to detect the cecal and gastric lesions on radiological examination, despite the indulgence of a highly competent radiologist and an excellent series of films. Perhaps the fluoroscopist's knowledge of a digitally palpable rectal lesion had caused him to focus his examination largely upon the left colon alone. Nevertheless, one should always bear in mind that roentgen examination of the colon, even in the very best of hands, is by no means infallible.[8] Of note, also, is the fact that on closer reexamination of these preoperative films after surgery had been carried out, the radiologists felt that the small gastric lesion might have been detected preoperatively but emphasized that this was purely retrospective, the defect by x-ray study being very equivocal.

SUMMARY

The case of a 64-year-old white man is presented in whom simultaneous primary carcinomas were found in the stomach, cecum, and rectum, each of which were operable. This is believed to be the first such case reported in which these three particular sites were involved by simultaneously occurring primary carcinomas. All of the tumors were successfully resected, those in the stomach and cecum being excised at one operation; this is the third such operative procedure reported. The patient remains apparently free of tumor 25 months postoperatively, although the most recent complete tumor survey carried out upon him was 14 months following surgery.

Simultaneous multiple primary malignant tumors, involving colon and stomach especially, are briefly discussed. When surgical exploration of the abdomen is carried out for a known gastrointestinal malignant lesion, careful examination of at least the stomach and entire colon for other primary tumors is imperative.

Dr. C. E. Gordon prepared the photomicrographs of the tumors.

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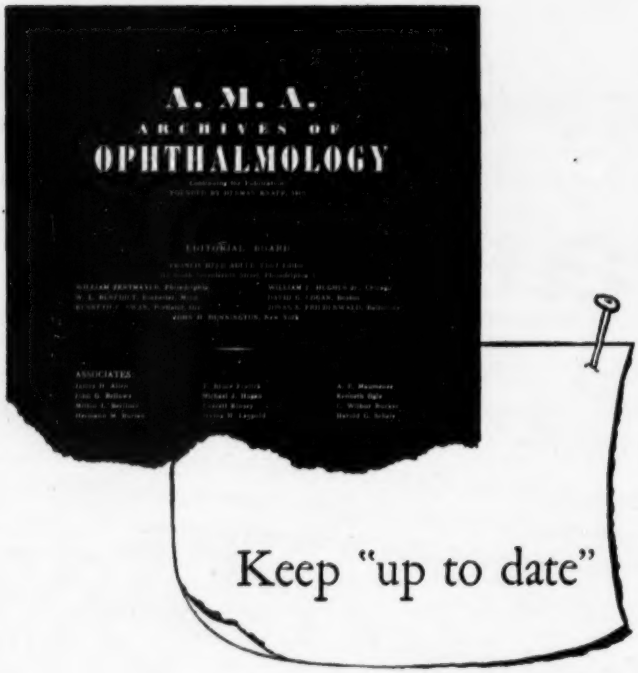

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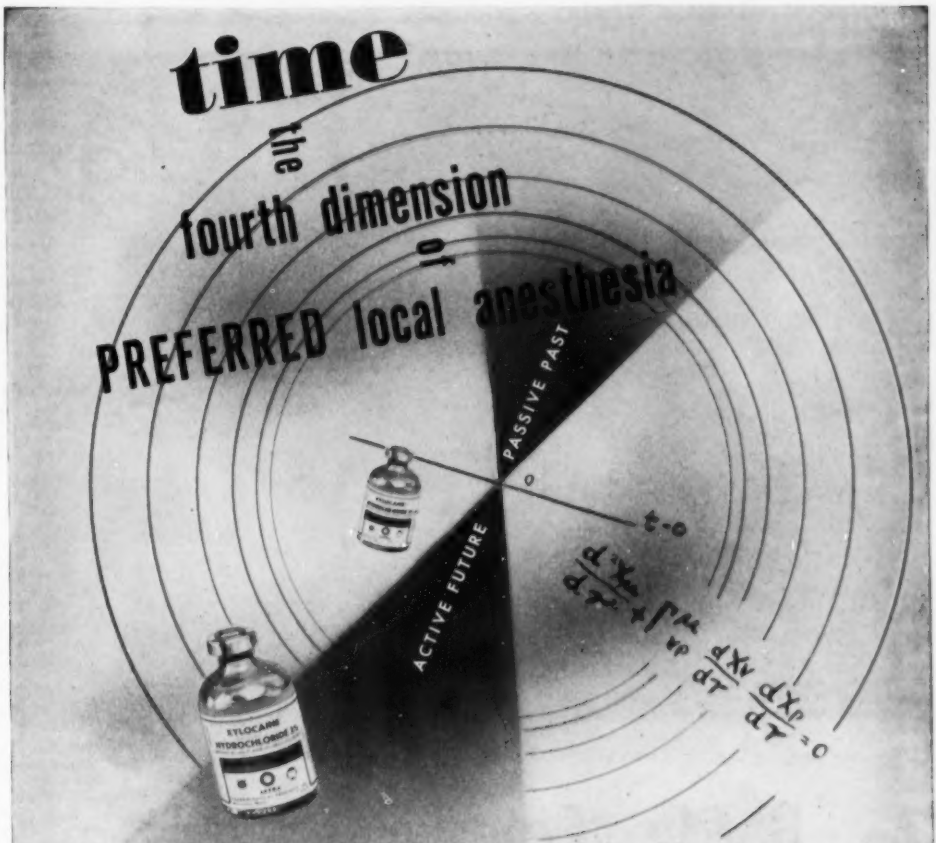

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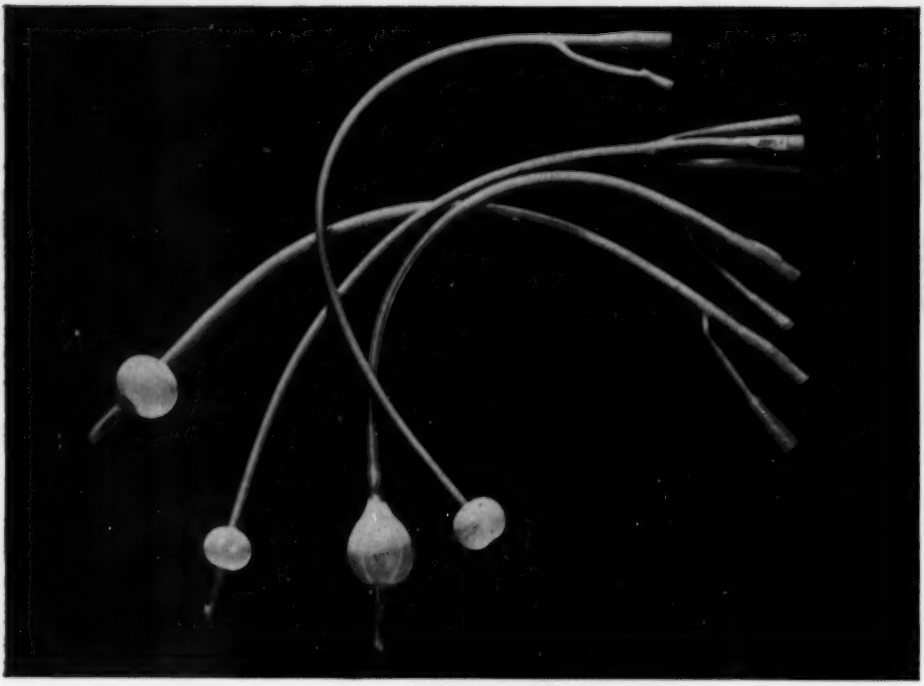

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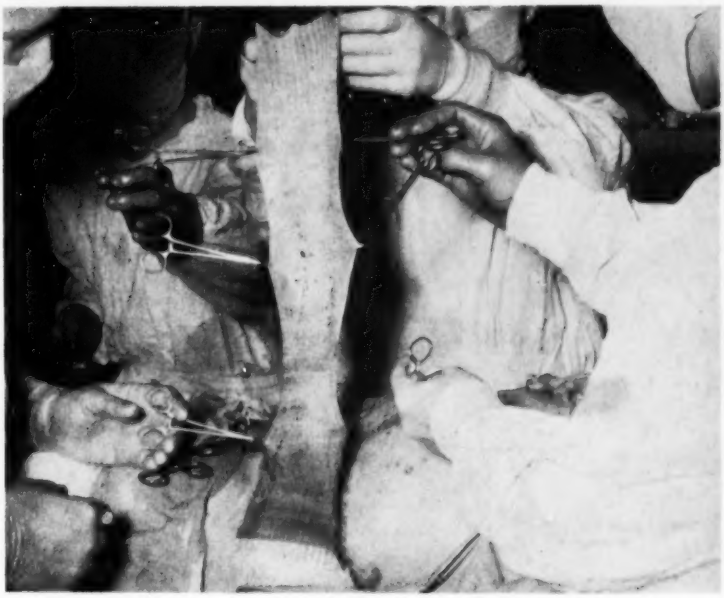

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